South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde

P.O. Box 643, Cape Town Posbus 643, Kaapstad

Cape Town, 20 October 1956 — Weekly 2s. 6d. | Vol. 30 No. 42 | Kaapstad, 20 Oktober 1956 — Weekliks 2s. 6d.

CONTENTS : INHOUD

South African Medical Journal
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Cape Town, 20 October 1956 Vol. 30 No. 42 Kaapstad, 20 Oktober 1956
Weekly 2s. 6d. Weekliks 2s. 6d.

SYPHILITIC COLD HAEMOGLOBINURIA

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The association of haemoglobinuria with syphilis has been known since 1885; the presence of the responsible haemolysin was first described by Donath and Landsteiner in 1904. However, the disease is an extremely rare one in spite of the fact that syphilis is so common. We have surveyed the literature for the past 10 years but have been unable to find any reference to the condition in the Bantu. Suckling (1951) described the disease in a Coloured boy aged 6 years who showed evidence of congenital syphilis.

The present case, occurring in a Bantu male, presented an interesting problem in that at first sight the condition appeared to be one of nocturnal haemoglobinuria.

CASE HISTORY

A.M., a Bantu male aged 32, was admitted to hospital with a history of having passed dark-coloured urine on getting up each morning. This had occurred each day for 7 days. The specimens passed during the rest of the day had been normal in colour. Apart from occasional attacks of vague abdominal pain, which had also been present for 1 week, he felt quite well. Enquiry into his past illnesses revealed that he had had a similar attack about 10 months previously, when he had passed dark-coloured urine for three or four days. He could not recall whether these specimens had been passed only first thing in the morning or during the rest of the day as well.

On Examination: He was a thin man with a normal temperature and pulse rate. The blood pressure was 130/90 mm. Hg. There was a faint icteric tinge to the conjunctivae. The abdomen was soft and no tenderness was elicited. The liver was palpable one finger-breadth below the costal margin. Examination of the other body-systems was essentially negative.

Urine: On admission the patient brought a specimen of urine which had been passed that morning. It was of a dark reddish-brown colour and spectroscopic examination showed large amounts of oxyhaemoglobin. No intact cells were seen in the spun deposit. No sugar was present. Tests for albumin were strongly positive.

Blood: The haemoglobin level on admission was 11·2 g.% (75%). The white-cell count was 8,500 with a normal differential count. The red cells showed marked polychromasia and the reticulocyte count was 10%. The plasma was icteric and contained oxyhaemoglobin. Schumm's test was positive. The blood Wassermann-reaction was strongly positive (40 dilutions with the Kolmer technique). The Donath-Landsteiner reaction was strongly positive, as was a direct Coombs test performed at room temperature. Estimation of cold haemagglutinins in the patient's serum gave a positive result at a titre of 1 : 16. The red-cell fragility test gave a normal result. On X-ray examination of the chest the appearance was normal. Intravenous pyelograms showed that both kidneys excreted the dye normally.

Progress and Treatment: The patient did not pass any further specimens of urine containing haemoglobin during his stay in hospital and examination of his serum after 3 days did not show any methaemalbumin, which disappears fairly rapidly in the absence of further haemolysis.

After 2 weeks in hospital the patient was treated with penicillin, being given a course of 9,000,000 units over a week. His haemoglobin level on discharge was 14·5 g.% (98%), and the P.C.V. was 43% and M.C.H.C. 34%. The urine was normal and the plasma no longer icteric.

DISCUSSION

In paroxysmal cold haemoglobinuria intravascular haemolysis is precipitated by exposure to cold.

In vitro the haemolysin becomes adsorbed onto red cells in the cold and, in the presence of complement, causes haemolysis of the cells when the temperature is raised to 37·0°C. Haemolysis will take place at a lower temperature with an active haemolysin. Mackenzie (1929) reviewed the literature on the condition and showed that about 95% of all cases of haemoglobinuria where this so-called 'warm-cold' haemolysin was present were due to syphilis.

Stats and Wassermann (1943) and Stats, Wassermann and Rosenthal (1948) distinguished another type of cold haemoglobinuria. This occurred in non-syphilitic patients and was associated with the presence of high levels of cold haemagglutinins in the serum. Cold haemagglutinins of this nature have been repeatedly demonstrated in atypical pneumonia and occasionally in other disease states. Dacie (1950), however, showed that a haemolysin might also be present in such cases along with the cold haemagglutinins and that the

haemolysin had certain features in common with that described by Donath and Landsteiner. In their review on cold haemagglutinins, Stats and Wassermann (1943) showed that these are not usually present in syphilitic cold haemoglobinuria where the Donath-Landsteiner antibody is present. Since then, however, Siebens, Zinkham and Wagley (1948) have reported that cold haemagglutinins may be present in high titre in such cases. It now appears that the differentiation of the two groups of haemoglobinuria—the syphilitic and non-syphilitic—may be somewhat artificial in respect of the type of antibody involved in the reaction.

Differential diagnosis of the condition involves a consideration of the other causes of haemoglobinuria; e.g. the nocturnal type (Marchiafava-Mitcheli syndrome), haemoglobinuria of exertion, and the allergic type of haemoglobinuria such as that resulting from sensitivity to the bean *Vicia fava*. Other causes, which are usually easily distinguished, include malaria, blackwater fever, incompatible blood transfusion, severe infections with certain organisms, especially of the *Clostridia* group, and various chemical poisons such as arseniuretted hydrogen. In most of the usual cases of haemolytic anaemia, however, the degree of haemolysis is not sufficient to result in haemoglobinuria, the threshold level for which is about 130-150 mg.%.

Syphilitic cold haemoglobinuria is commoner in males than in females, probably because of the greater incidence of syphilis in males. It may affect those suffering from congenital syphilis as well as those with an acquired infection.

The ease with which haemoglobinuria may be precipitated by exposure to cold varies in different patients. In some the exposure has to be prolonged whilst in others the mere immersion of the hands in cold water appears to be sufficient to cause an attack. It seems, however, that other factors are involved since exposure to cold will not invariably result in haemolysis with haemoglobinuria. Some patients who have the Donath-Landsteiner type of antibody in their serum never suffer from haemoglobinuria.

The clinical picture varies considerably. In some patients there are no constitutional upsets during or after an attack. In others, there may be pain in the back and limbs, with diarrhoea and abdominal pain. Some cases show the features of Raynaud's syndrome in addition, but haemoglobinuria may be associated with Raynaud's syndrome in the absence of any evidence of syphilis.

The present case illustrates one point of importance in the differential diagnosis. The patient's history that he passed dark-coloured urine only on getting up in the morning led us to suspect that he was suffering from the nocturnal type of haemoglobinuria. The explanation for this lies in the fact that during the week in question the night temperatures were very low and the patient had been sleeping with very little in the way of covering. His previous attack 10 months before had also occurred during very cold weather.

In view of the presence of a positive Wassermann reaction and a positive Donath-Landsteiner test we did not feel justified in trying to precipitate an attack of haemolysis by such methods as the immersion of limbs in cold water. This is a dangerous procedure and may lead to renal anoxia and fatal uraemia.

SUMMARY

The case history of a patient with paroxysmal syphilitic cold haemoglobinuria is discussed. This case is of particular interest in that the history suggested that the patient was suffering from nocturnal haemoglobinuria.

We are indebted to Dr. R. E. Stevenson, Director Provincial Medical and Health Services, for permission to publish this paper and Dr. W. Phillips for access to the case notes.

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Suid-Afrikaanse Tydskrif vir Geneeskunde
South African Medical Journal

EDITORIAL

EXTENSIVE RESECTION OF THE SMALL INTESTINE

Most, perhaps all, organs of the body have a considerable reserve in hand. Thus destruction of some nine-tenths of liver substance is necessary before hepatic function is disturbed. The small bowel's reserve is not so great as this, but still is quite large, and it would seem that two-thirds of its total length may be removed without any untoward symptoms or even biochemical evidence of malabsorption becoming evident. Even when much greater quantities are removed the end-result is not necessarily disastrous, and in recent years there have been several examples of good health in patients who recovered with as little as 18 inches of jejunum and ileum remaining. In this country the patient Toni suffered removal of all but 6-7 inches of jejunum and ileum following a complicated intra-abdominal condition which included both mesenteric thrombosis and volvulus.[1] Despite complete lack of care in his home management, he lived for 3½ years before dying in a state of inanition.[2]

It is thus pleasing to be able to tell the surgeon, who may be faced with large tracts of gangrenous gut, that he may expect good results from quite massive resections. The literature is somewhat puzzling because of several accounts of patients who did badly after quite small resections. In all of these cases, however, there was some other cause for malabsorption, such as a spreading chronic enteritis (Crohn's disease) or the presence of a loop or by-passed section of small bowel. It is not generally appreciated that a sprue-like syndrome may be produced when a segment of small gut is removed from the main circuit. The cause of this so-called 'loop syndrome' is uncertain, but it appears to be related to stagnation in the segment involved. In the case of a gastrocolic fistula, for example, the malnutrition which results is an example of this syndrome and is not caused through a loss of absorbing surface, since in fact most of the ingested food goes along the normal channel and not through the fistula. The advice may therefore be given to the surgeon in operating on a case of Crohn's disease, for instance, not to be afraid of making a

VAN DIE REDAKSIE

GROOTSKAALSE RESEKSIE VAN DIE DUNDERM

Meeste, miskien alle, organe van die liggaam het 'n aansienlike reserwe in voorraad. Dus is vernietiging van sowat nege-tiendes lewerstof nodig voordat die funksie van die lewer verstoor word. Die dunderm se reserwe, alhoewel nie so groot nie, is nogtans heeltemal groot en dit lyk of twee-derdes van sy totale lengte verwyder kan word sonder dat daar enige teenspoedige simptome of selfs biochemiese bewys van gebrekkige absorbering voorkom. Selfs wanneer baie groter stukke verwyder word, is die endresultaat nie noodwendig noodlottig nie, en gedurende die afgelope jare was daar verskeie voorbeelde van goeie gesondheid by pasiënte wat herstel het, al het daar net sowat 18 duim van die nugtere derm en kronkelderm oorgebly. In ons land het die pasiënt Toni verwydering van sy nugtere derm en kronkelderm tot op 6-7 duim ná ondergaan, as gevolg van 'n ingewikkelde binnebuikse toestand waar beide trombose van die dermhangband en 'n dermknoop[1] aanwesig was. Ten spyte van 'n totale afwesigheid van sorg in sy huislike omstandighede, het hy nog 3½ jaar geleef voordat hy in 'n toestand van uittering gesterf het.[2]

Dit is dus aangenaam om in staat te wees om die chirurg, wat te kampe mag hê met groot stringe gangreneuse derm, mee te deel dat hy goeie resultate van heel massiewe reseksies kan verwag. Die literatuur is ietwat verwarrend vanweë verskeie verslae van pasiënte wat nie so goed gevaar het na heel klein reseksies nie. By al hierdie gevalle egter was daar 'n ander rede vir gebrekkige absorbering, soos byvoorbeeld 'n spreiende kroniese dermontsteking (Crohn se siekte), of daar was 'n lus of uitgeslote seksie van die dunderm aanwesig. Dit word nie algemeen besef dat 'n spru-agtige sindroom veroorsaak mag word nie as 'n deel van die dunderm van die hoofkanaal afgeskei word. Die oorsaak van hierdie sogenaamde ‚lussindroom' is onseker, maar dit blyk verwant te wees aan stilstand in die betrokke deel. In die geval van 'n maag-dikdermfistel, byvoorbeeld, is die ondervoeding wat daarop volg 'n voorbeeld van hierdie sindroom en word dit nie veroorsaak deur verlies van absorberings-oppervlakte nie, aangesien die meeste voedsel wat ingeneem word, in werklikheid deur die normale kanaal en nie deur die fistel gaan nie. Die advies kan dus aan die chirurg gegee word wanneer

resection radical enough to eliminate all possible areas involved, and not to fashion by-passes, loops or pouches.

Two rather strange uses for the operation of massive intestinal resection have been put forward. Since several patients after this operation lost weight, but otherwise remained free of symptoms, Henrikson actually excised part of the small bowel in order to correct obesity, but the result was not altogether satisfactory, since his patient lost too much weight. Kremen *et al.*[3] claimed that removal of the distal half of the small bowel in dogs caused defective fat-absorption and loss of weight, whereas no such effect occurred after resection of the proximal half. More cunning than Henrikson, he proceeded then to exclude, rather than to excise, all but the terminal 18 inches of the lower half of the small bowel. The excluded loop was made to open distally into the transverse colon and was to be kept in storage, so that if too much weight were lost some of it might be returned to the main intestinal pathway later. Neither the author of this surgical extravaganza nor the discussers of his paper nor the commentator in the *Lancet*[4] noticed what fine conditions obtained for the development of the stagnating-loop syndrome.

The second use for intestinal resection is in the treatment of ascites caused by portal hypertension. Bernhard and co-workers[5] argued that the object of portacaval shunt was to reduce the flow of blood through the portal system. Now the great majority of that blood passed through the mesenteric vascular bed associated with the small intestine. Resection of a large portion of small bowel, with its vascular bed, should therefore greatly reduce the portal blood-flow, and might slow up or even prevent the formation of ascites. To test this idea, ascites was produced in dogs by constriction of the supradiaphragmatic inferior vena cava, and the effects on the re-accumulation of this ascites after various types of resection were observed. Removal of 80% of small gut completely prevented the formation of ascites, and the animals were reputed to regain good health.

Despite this good result, there has been no spate of similar operations performed in humans, although Fuller,[6] in 1937, had actually forestalled Bernhard and reported successful diminution of ascites in a cirrhotic patient after resection of only 6 feet 8 inches.

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hy byvoorbeeld op 'n geval van Crohn se siekte opereer, om nie bang te wees om 'n reseksie te doen wat radikaal genoeg is om alle moontlike aangetaste dele te verwyder nie, en om nie omlooppaaie, lusse of sakke te vorm nie.

Twee enigsins eienaardige gebruike van die operasie vir massiewe ingewandsreseksie is aan die hand gedoen. Aangesien verskeie pasiënte na hierdie operasie gewig verloor het, maar andersins vry van simptome gebly het, het Henrikson, ten einde vetsugtigheid te bestry, inderdaad 'n deel van die dunderm uitgesny, maar die resultaat was nie heeltemal bevredigend nie, aangesien sy pasiënt té veel gewig verloor het. Kremen *et al.*[3] het daarop aanspraak gemaak dat verwydering van die distale helfte van die dunderm by honde gebrekkige vetabsorbering en gewigsverlies veroorsaak terwyl dit nie op reseksie van die proksimale helfte volg nie. Slimmer as Henrikson, het hy toe voortgegaan om die laer helfte van die dunderm tot op die laaste 18 duim ná, af te sluit, liewers as om dit uit te sny. Hy het die distale end van die afgeslote lus in die dwars-dikderm laat open—dit moes in bewaring gehou word sodat, indien té veel gewig verloor was, 'n deel daarvan later na die hoofdermkanaal teruggebring kon word. Nóg die skepper van hierdie chirurgiese extravaganza, nóg die besprekers van sy verhandeling, nóg die kommentator in die *Lancet*[4] het opgemerk watter suiwere toestande verkry is vir die ontwikkeling van die stilstaanlus-sindroom.

Dermreseksie word tweedens gebruik by die behandeling van buikwatersug veroorsaak deur poortdrukverhoging. Bernhard en sy medewerkers[5] het geredeneer dat dit die doel van poortholaarverbinding is om die vloei van bloed deur die poortstelsel te verminder. Wel, die grootste hoeveelheid van daardie bloed het deur die vatbed van die dermhangband gegaan wat met die dunderm verbind is. Reseksie van 'n groot deel van die dunderm, met sy vatbed, behoort dus die poort-bloedstroom te verminder en mag die vorming van buikwatersug vertraag, of selfs verhoed. Om hierdie idee te toets, is buikwatersug by honde veroorsaak deur vernouing van die onder-holaar bokant die middelrif, en die gevolge wat verskillende tipes van reseksie op die hervorming van hierdie buikwatersug gehad het, was opgemerk. Verwydering van 80% van die dunderm het die vorming van buikwatersug heeltemal verhoed, en dit is beweer dat die diere goeie gesondheid herwin het.

Ten spyte van hierdie goeie resultaat, was daar geen vloed van soortgelyke operasies wat op mense uitgevoer is nie, alhoewel Fuller[6] in 1937 inderdaad vir Bernhard voorgespring het en 'n suksesvolle vermindering van buikwatersug by 'n sirrotiese pasiënt, na reseksie van slegs 6 voet 8 duim, gerapporteer het.

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FRACTURES OF THE NECK OF THE FEMUR*

A METHOD OF ASSESSING THE VIABILITY OF THE FEMORAL HEAD

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The blood supply of the head of the femur is precarious. This fact is manifested by the frequency with which trauma to the hip joint is followed by partial or total avascular necrosis of the femoral head. In the management of traumatic lesions around the hip joint, therefore, a thorough knowledge of the blood supply of the region is essential, and in this regard valuable contributions have been made recently by Wolcott (1943), Tucker (1949), Trueta and Harrison (1953) and Judet (1955).

Impressed by the need to establish some method of assessing the vascularity of the head of the femur in fractures of the femoral neck we reviewed some of the previous work on the subject, paying particular attention to the intra-osseous circulation. In cadavers the vessels

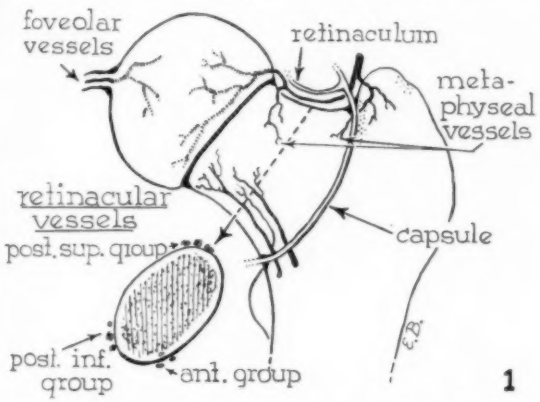

Fig. 1. Diagram to show blood supply to the head of the femur. This is an anterior view of the neck of the femur showing how the vessels run up the retinaculae to the head. In this position they are very vulnerable when the neck of the femur is fractured.

under study were outlined by the intra-arterial injection of India ink. The upper third of the femur was then excised and cleared, a modification of the Spalteholtz technique being used. The blood supply of the gross specimens could then be clearly outlined. The intra-osseous circulation was studied by dividing the femoral head into 2 millimetre slabs so that the vessels could be followed with the dissecting microscope. Our results were in agreement with the findings of Tucker (1949) and can be summarized as follows (Fig. 1):

1. *The foveolar artery* arises from the obturator artery and passes underneath the transverse ligament and along the ligamentum teres. It may arise on occasion from the medial circumflex artery. In children this vessel does not supply a significant part of the head; in fact, it supplies the ossification centre in less than 50% of cases. In adults, on the other hand, the vessel is larger and anastomoses with the metaphyseal vessels that have penetrated the site of the old epiphyseal plate.

2. *The retinacular arteries* pierce the capsule close to the femur and course along the femoral neck with the reflected part of the capsule. They are arranged in 3 groups; postero-superior, postero-inferior, and anterior. The first 2 groups are the larger and the more constant. They originate from the medial circumflex artery. The anterior group is the smallest and is derived from the lateral circumflex artery.

These vessels send branches to the superior part of the femoral head and the neck of the femur, and in addition they anastamose with the nutrient artery of the femoral shaft in adults. There is no anastomosis between the nutrient artery and the arteries in the femoral head in children prior to fusion of the epiphyseal plates, and even in adults there is no evidence so far to show that the anastomosis is sufficient to keep the head alive when other sources of blood supply are damaged.

The venous return follows much the same pattern as the arterial supply (Hulth, 1953).

There are many causes of avascular necrosis (Table I). After *fractures* of the femoral neck the retinacular vessels may be ruptured or occluded by post-traumatic thrombus formation. The endosteal circulation from the nutrient anastomosis is, of course, interrupted by the fracture. The foveolar artery usually remains intact. Survival of the head of the femur after fractures of the femoral

TABLE I. CAUSES OF AVASCULAR NECROSIS OF THE FEMORAL HEAD

Traumatic

1. Fractures of the neck of the femur—especially sub-capital fractures
2. Dislocation of the hip—especially central dislocations
3. Sub-capital osteotomy for slipped femoral epiphysis
4. Forceful manipulation to reduce congenital dislocation of the hip

Non-Traumatic

1. Caisson disease
2. Perthes' disease
3. X-ray necrosis of the head of the femur

TABLE II. INCIDENCE OF AVASCULAR NECROSIS OF THE HEAD OF THE FEMUR FOLLOWING FRACTURES OF THE FEMORAL NECK

Christophe (1953)	15% (in 104 cases)
Wardle (1949)	30% (in 46 cases)
Bado (1948)	25% (in 202 cases)
Linton (1944)	40%
Dickson (1953)	25%

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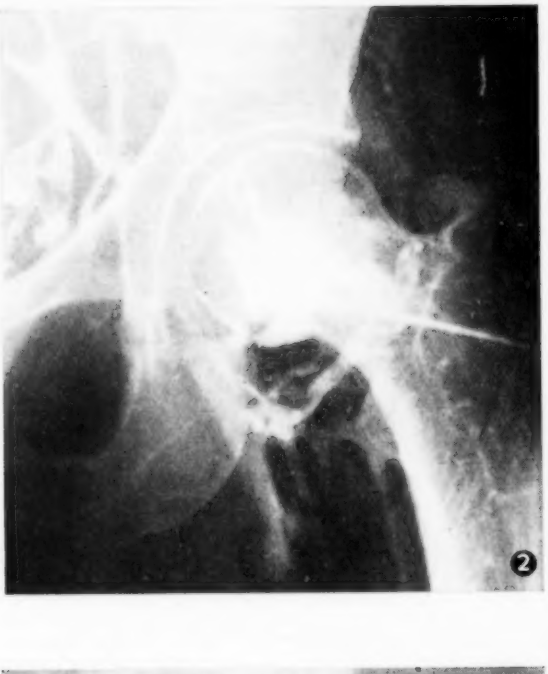

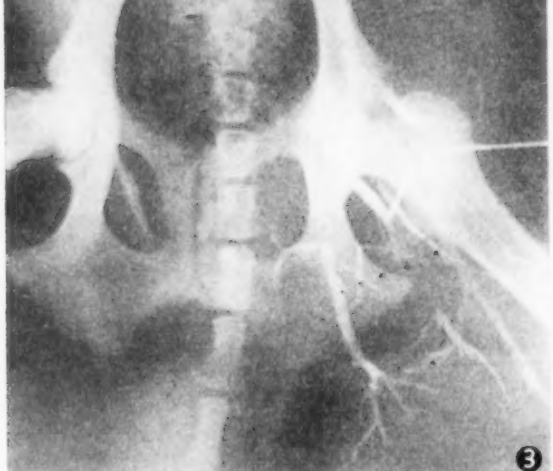

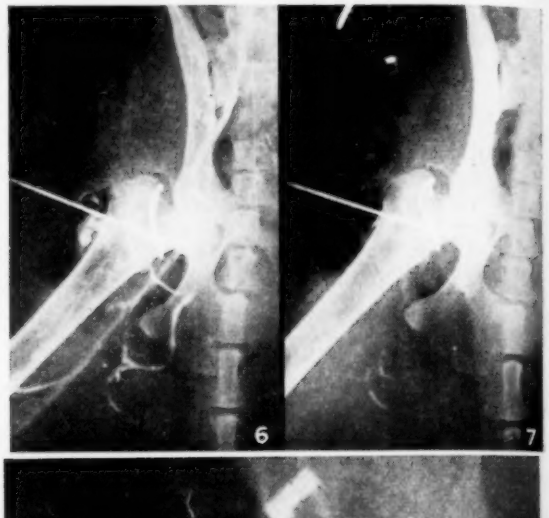

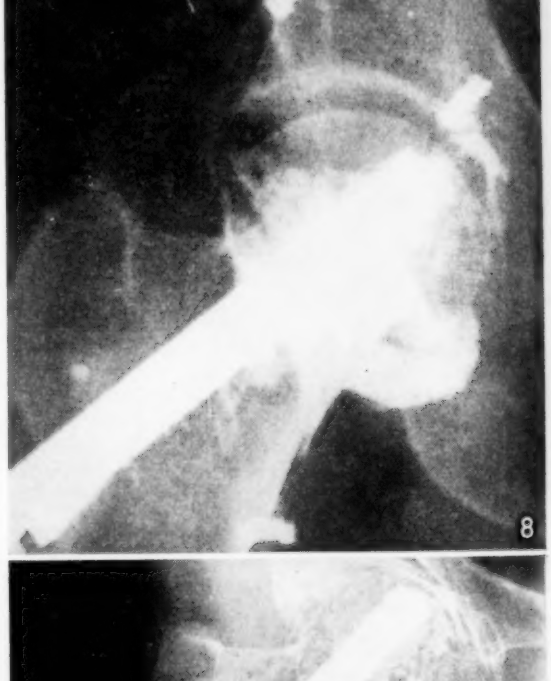

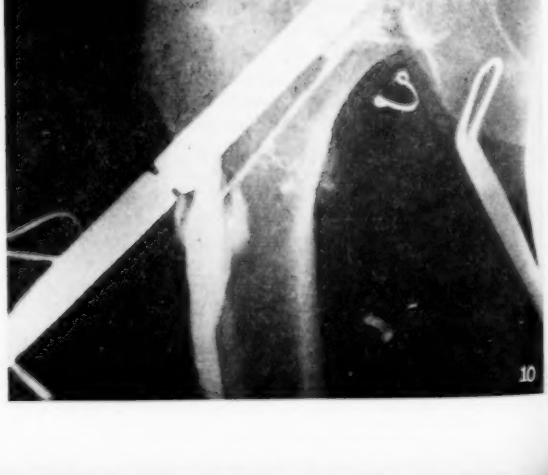

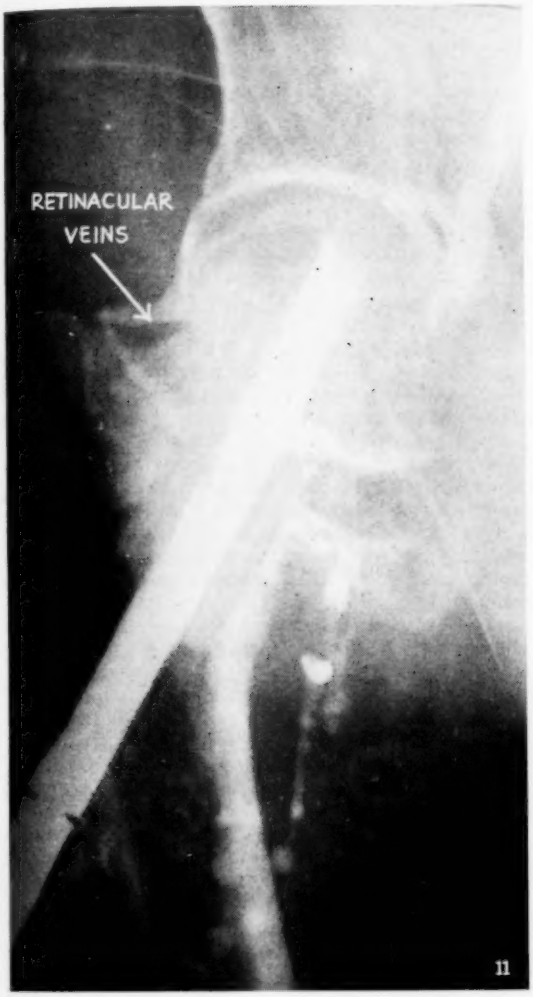

Fig. 2. Positive phlebogram obtained when the head of the femur is injected with sodium iodide in a cadaver.

Fig. 3. Positive phlebogram obtained when the neck of a femur of a dog is injected with sodium iodide.

Fig. 4. A sub-capital osteotomy has been performed in a manner to simulate a trans-cervical fracture of the neck with disruption of the retinacular vessels. Sodium iodide was injected into the head of the femur. Because the venous drainage of the head had been interrupted there was no filling of the venous tree.

Fig. 5. Filling the capsule of the hip joint with sodium iodide. Note the radio-opaque substance is absorbed so slowly that a positive phlebogram does not result.

Fig. 6. In this dog a positive phlebogram was obtained after injecting sodium iodide into the head of the femur even though the retinacular vessels had been occluded with a circular loop of wire. On studying this X-ray closely it can be seen that the nutrient system of the femur had been filled with dye and the dye is being absorbed from the shaft of the femur. This experiment is of significance in the assessment of clinical phlebograms in which dye escapes into the distal fragment.

Fig. 7. This X-ray was taken 2 minutes after the X-ray shown in Fig. 6 and demonstrates the rapidity with which the radio-opaque substance is absorbed from the bone and leaves the venous tree.

Fig. 8. Typical radiological appearance of a negative phlebogram. The Diodrast is seen to remain in the head and a collar of dye is formed round the fracture site due to leakage into the fracture haematoma. This leakage does not seem to occur if there is an intact venous return.

Fig. 10. This X-ray was taken in a clinical case in which two guide-wires had been used. Notice how the Diodrast has escaped down the track of the discarded wire into the distal fragment. In all cases in which dye was seen in the distal fragment no conclusion could be reached as to whether the dye was being absorbed from the head of the femur or from the distal fragment.

Fig. 11. Positive phlebogram following injection of Diodrast into the head of the femur at the time of nailing. Notice the circumflex veins and the retinacular veins. Although some dye can be seen in the distal fragment this phlebogram was taken as a true positive because the retinacular veins had been outlined.

Fig. 12. The 15-minute film showing that the dye has been absorbed from the head of the femur.

neck depends on the amount of damage that has been sustained by the retinacular vessels at the time of injury and during the subsequent handling of the patient. The figures quoted in the literature on the incidence of avascular necrosis (Table II) vary from 15% to 40%. Although these figures show an alarmingly high incidence of avascular necrosis they probably all under-estimate the incidence of this complication, for the following reasons.

1. Death of the femoral head may occur without producing any radiological changes. The classical sign of a dense femoral head may never be seen.

2. The fracture may unite in the presence of avascular necrosis and the patient may be able to start painless weight-bearing. The first time the clinician finds out that avascular necrosis occurred may be 2-3 years later when, because of increasing pain in the hip, a further X-ray is taken which reveals a collapsed, deformed, femoral head.

Dewar and his co-workers (1956) have stated that if the long-term results of fractures of femoral necks are studied, it is found that the incidence of avascular necrosis is about 50%.

Re-vascularization of the dead head can and does occur (Phemister, 1949). If the process of re-vascularization was constant, rapid and complete, the problem of management of these cases would not be so great. Unfortunately this is not so. Histological studies we undertook showed that occasionally the process was completed in 3 or 4 months, but this was the exception. Much more frequently we found that even after 18 months the process of re-vascularization was not complete. Dewar and Simurda (1956), using radio-active phosphorus, showed that the re-vascularization process follows a certain pattern and that unfortunately the last portion of the head to form new bone is the weight-bearing area. In order to prevent crushing of the femoral head in patients suffering from avascular necrosis it would be necessary therefore to prevent all weight-bearing until the re-vascularization process has been completed. Management of this apparently simple fracture is further complicated by the fact that this re-vascularization process may produce a dense head on X-ray. This is not a relative density but is a *real* density due to appositional new-bone formation. Twelve femoral heads that had been excised on the basis of the radiological diagnosis of avascular necrosis were examined histologically. Only 2 were found to be entirely necrotic—8 showed re-vascularization with new-bone formation in varying degree, and 2 showed complete re-vascularization of the head!

Whether the surgeons decide to treat avascular necrosis conservatively by preventing weight bearing or operatively by replacing the dead head with an endoprosthesis, the management of these cases would be greatly simplified if some method were available to assess the vascularity of the head of the femur at the time the initial treatment is carried out. Rook (1953) attempted to do this by means of arteriograms, but by this method it was difficult to outline the retinacular vessels and impossible to see the intra-osseous circulation. Tucker described the use of radio-active phosphorus. Radio-active phosphorus was injected intravenously and the radio-activity of the femoral head was then assessed by means of a needle Geiger-counter. However, Tovee, Dewar and Simurda (1956) showed that owing to ionic jump this method was inaccurate, since even a dead head could pick up radio-activity.

EXPERIMENTAL STUDY

In view of the failure of these previously described methods it was decided to attempt to assess the vascularity of the head of the femur in recent fractures by studying its venous drainage. The venous drainage can be outlined by injecting a radio-opaque substance into the intertrabecular spaces of the cancellous bone of the head. Since the veins draining the head of the femur accompany the arteries, it may be assumed that a positive phlebogram indicates an intact arterial supply. If the venous drainage of the head has been destroyed it would not be possible to obtain a phlebogram. In such instances, when all venous drainage has been cut off, the femoral head would die even if the arterial supply has miraculously escaped damage (Hulth, 1953).

The method was studied experimentally at first on cadavers and dogs. A lumbar-puncture needle was introduced into the shaft of the femur of a cadaver just below the great trochanter and pushed up into the head of the femur; 10 c.c. of a 50% solution of sodium iodide was then injected and an X-ray taken instantaneously. This showed in great detail the veins draining the upper end of the femur (Fig. 2). The other hip joint was then exposed and an osteotomy of the neck of the femur was performed, simulating a sub-capital fracture. On injecting the femoral head with sodium iodide we found that the dye remained in the head. The veins were not outlined. These findings may be criticized on the grounds that the veins in a cadaver are clotted and that a negative phlebogram does not necessarily mean a disrupted venous return. The experiment was therefore repeated on dogs. Injection of a normal dog's hip produced a positive phlebogram (Fig. 3). After sub-capital osteotomy with destruction of the retinacular vessels the injection gave a negative result (Fig. 4). After mid-cervical osteotomy, on the other hand, when the retinacular vessels were preserved, injection of the femoral head gave a positive phlebogram.

Various radio-opaque substances were employed and the general and local reactions were studied. Sodium iodide gave rise to convulsions and was therefore not applicable in clinical cases. Thorotrast produced necrosis, but 35% Diodrast did not produce any local necrosis of cancellous bone.

From the experimental evidence it would appear that it was safe to use Diodrast as a contrast medium and that by injecting this substance into the head of the femur it was possible to demonstrate whether the venous return was intact. A positive phlebogram indicated that the head retained its normal vascular channels whereas a negative phlebogram indicated that the venous return had been seriously interfered with. But before applying this method clinically certain sources of error had to be investigated. It might be argued that the dye could escape *via* the fractured surface and be absorbed from the capsule of the joint. If this were possible then the

venous tree would be outlined even when the head of the femur had been severed from all its vascular connections. To investigate this possibility the capsule of the hip joint of a dog was filled with dye. Absorption was so slow that the venous tree was not outlined (Fig. 5). The results of this experiment are in accord with our clinical experience. For example, arthrograms of the hip or knee performed clinically never show venous filling. We feel confident therefore that leakage of the dye into the capsule could not account for the positive phlebograms that were obtained.

If the radio-opaque substance injected into the head of the femur leaked into the distal fragment, absorption from the distal fragment might give rise to a positive phlebogram. This was tested experimentally. The retinacular vessels of the neck of the femur in a dog were occluded and a radio-opaque substance was injected into the femoral head. A positive phlebogram was obtained (Figs. 6 and 7). This fact has to be borne in mind when analysing the clinical results. It is possible for dye to leak from the head into the distal fragment under the following circumstances: (1) Very firm impaction of the fracture with dye leaking across the impacted inter-trabecular spaces. (2) If more than one guide-wire is used and the tips of the two wires are in close proximity, then when dye is injected it is possible for the dye to leak back down the track of one of the wires into the distal fragment (Fig. 10).

It was felt therefore that any case that showed dye in the distal fragment had to be excluded from the series because there was no means of telling whether absorption had taken place from the head of the femur or from the distal fragment. Similarly if one of the guide-wires is introduced too far and enters the acetabulum, it is possible for some of the dye to escape through the joint into the acetabular roof and be absorbed from the innominate bone. In such cases also no conclusions can be drawn from the X-ray picture.

CLINICAL APPLICATION

As we have said previously, no positive statement can be made on the incidence of avascular necrosis as diagnosed by clinical and radiological means unless cases have been followed for at least 3 years. In studying the usefulness of this method some technique had to be devised that did not interfere with the normal method of reducing and fixing fractures of the femoral neck. It was decided therefore to treat the cases by reduction and fixation of the fragments with a Smith-Petersen nail and to inject the head of the femur by dye injected up the centre canal of the nail. A special adaptor was designed to fit into the end of the Smith-Petersen nail to facilitate the injection of dye (Fig. 9). With the nail in position 10 c.c. of 35% Diodrast (70% Diodrast gives a better picture) is injected. As the last c.c. is being injected an X-ray is taken. In those cases showing adequate venous drainage of the head a positive phlebogram will be demonstrated at this time. A further X-ray is taken after the lapse of 15 minutes. This film is of great use in those cases where there is some doubt whether the venous drainage of the head has been outlined. If the 15-minute film shows that all the dye has been absorbed from the head one can conclude that there must have been fairly adequate venous drainage from the head. On the other hand, if the 15-minute film shows dye still remaining in the head then this is further corroboration of the fact that the venous return has been seriously interfered with.

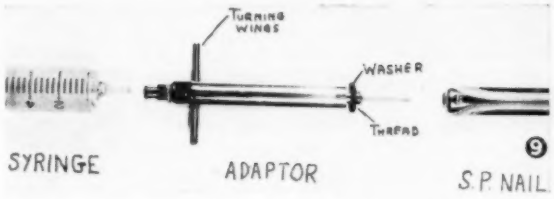

Fig. 9. Photograph showing the adaptor which was used to facilitate the injection of radio-opaque material in clinical cases. The adaptor screws into the Smith-Petersen nail. Latterly we have been using a nylon syringe to obviate the danger of breakage of the nozzle. Occasionally considerable force has to be used to inject the radio-opaque material into the head of the femur.

The technique has been used in 22 cases to date. In 8 cases we have unfortunately had to exclude the results from our series because of various technical errors. In one of these the guide-wire had penetrated the joint cavity and the arthrogram that resulted obscured the radiological picture. In the other 7, although there was a varying degree of filling of the veins, dye was present in the distal fragment and it was not possible to tell whether the absorption of the dye had taken place from the femoral head or from the distal fragment. In one of these cases 2 guide-wires had been used and their tips had been in contact; when the nail was inserted and injection made, the dye followed the route of least resistance along the track of the discarded guide-wire (Fig. 10), resulting in a filling of the distal fragment.

Of the 14 remaining cases, 4 had a typical negative phlebogram—that is to say, filling of the capital fragment, a collar of radio-opaque material round the neck caused by leakage of the dye into the fracture haematoma, and complete lack of filling of the venous tree (Fig. 8)—and 10 showed filling of the venous tree in varying degree, resulting in 2 typical patterns of phlebogram:

1. Visible filling of the retinacular veins. This was most frequently seen along the upper border of the femoral neck. When the retinacular veins are filled it indicates without doubt that a vascular communication with the head of the femur exists. These films may therefore be considered as true positive phlebograms whether or not dye is seen in the distal fragments (Figs. 11 and 12).

2. Filling of the circumflex veins or profundis femoris vein in the absence of dye in the distal fragment. In these cases the only possible way in which the dye could have reached these veins is from the capital fragment.

Follow-up of Cases

This is a preliminary report. The method has only been employed for 1 year and final adjudication of the method will have to wait for the lapse of 2 or 3 years.

Of the 4 with negative phlebograms one case suffered collapse of the femoral head when weight bearing was

started. The patient was treated by a replacement arthroplasty and this gave us the opportunity of histological study of the femoral head, which showed total avascular necrosis with no signs of re-vascularization. In one case, 4 months after injury the fracture showed some displacement; radiologically the head showed increased density and the patient had pain in the hip on partial weight-bearing. The other 2 cases are as yet free from symptoms. One patient has started weight bearing and although the other patient is still in bed there is no radiological evidence of avascular necrosis to date. These 2 cases are being followed with interest.

On the 10 with 'true' positive phlebograms 6 cases have commenced weight bearing and are free from pain. The remaining 4 cases are still non-weight-bearing. In none of these cases is there any evidence of avascular necrosis.

CONCLUSION

This paper is published as a preliminary report on a method of assessing the vascularity of the head of the femur that is still under investigation. The method is simple and does not entail any special apparatus nor does it entail any change of technique. There is no risk to the patient provided his sensitivity to Diodrast is tested before injection. The report has been presented in this preliminary stage in the hope that other surgeons would test the method in their own cases. Thus a statistically significant number of cases might be assessed within a short time and the practical advantages of the method properly evaluated. The cases that will present difficulty are those in which the blood supply to the head of the femur has been grossly interfered with but not completely cut off. In such instances it is theoretically possible for the dye to enter the remaining vascular portion of the head and a positive phlebogram results. Only time and testing will demonstrate whether this does, in fact, occur.

SUMMARY

Avascular necrosis occurs relatively frequently after fractures of the femoral neck. The radiological diagnosis is not dependable and clinically the diagnosis is often made by the advent of complications. Knowledge of the state of the blood supply to the head of the femur early in treatment would greatly facilitate the management of these cases. A method for assessing the vascularity of the head of the femur has been described and a plea has been entered for its wider application.

This study was undertaken as a part of an investigation into the blood supply of bone under a grant from the National Research Council of Canada. We should like to acknowledge our indebtedness to the clinicians who have so kindly allowed this method to be tested on their cases and in particular Dr. W. White, who put the facilities of the fracture service of the Toronto Western Hospital at our disposal. We are also greatly indebted to Dr. W. Anderson for his help in the histological studies, to Miss Blackstock for the illustration, and to Mr. Harold Lane for the photography.

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RESPIRATORY INFECTIONS DUE TO THE APC GROUP OF VIRUSES (ADENOVIRUS)

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In 1953 Rowe and his associates[1] described the isolation of a new tissue-culture cytopathogenic agent, later named the adenoidal-pharyngeal-conjunctival (APC) virus, which they repeatedly isolated from human adenoid tissue undergoing spontaneous degeneration in tissue cultures. They noted at that time that further studies were in progress to determine the role of this agent in human disease, particularly upper respiratory infections. Since then Rowe and associates[2] have reported the isolation of 171 strains of viruses producing cytopathogenic effects in human embryo tissue culture or Hela cell (human epidermoid carcinoma) cultures. Of these viruses, 57 strains were isolated from cultures of adenoids and tonsils undergoing spontaneous degeneration, 61 from nasopharyngeal secretions, 35 from conjunctival swabs, 15 from anal swabs, and 3 from tissue suspension. Six distinct immunological types of virus were found to be represented in this collection. The viruses in this group as a whole had an almost identical cytopathogenic change produced in Hela cells, human embryonic epithelium and fibroblasts, and monkey kidney tissue cultures, and shared complement-fixing antigens. They were found to be ether resistant and to lack pathogenicity for laboratory animals. This latter property accounts for their escape from recognition until the application of the tissue-culture technique to their isolation and study.

APC viruses were found in the adenoids, tonsils, or both, of 57·4% of children whose tissues were cultured. Presumably these children had indications for removal of their tonsils and perhaps the incidence of the infection is less in children whose tonsils do not require removal. However, the figure of isolations is a minimum one, and would have been higher if more detailed studies had been carried out; it indicates clearly that these virus infections are very prevalent. It is therefore of interest to note that their role in causing human disease is becoming clearer. The work of Hilleman and his associates[3, 4] in particular has revealed that this group is an important cause of respiratory disease in Man. Recently they recovered a new agent from a patient with primary atypical pneumonia (PAP) in an epidemic of an acute respiratory illness, which occurred at Fort Leonard Wood. This agent multiplied in human cell tissue cultures, producing obvious cytopathogenic changes. It produced no disease in the common laboratory animals. The patient developed specific neutralizing and complement-fixing antibodies for the agent. Other patients in the epidemic with primary atypical pneumonia also developed these antibodies, but those patients whose illness was proved to be due to Fluvirus A[1] did not. Most of the cases in this epidemic presented evidence of infection with this recently discovered virus. Clinically the patients presented signs and symptoms of involvement of the respiratory tract. The onset of illness in most cases was relatively gradual and most patients were admitted to hospital on the second or third day of their disease. The acute symptoms were superimposed on minor complaints, such as non-productive cough, mild rhinitis, or mild hoarseness.

The predominant complaints on admission to hospital were pharyngitis, hoarseness, cough and chilliness. On examination it was noted that there was redness and swelling of the anterior and posterior tonsillar pillars, uvula and posterior pharyngeal wall. Most had a thin confluent mucopurulent discharge in the area of the pharynx. Most had a cough which produced mucopurulent sputum and was associated with substernal aching and pretracheal pain. Many, but not the majority, of patients complained of frontotemporal headache and also of myalgia. The average duration of fever after admission to hospital was 5·3 days in primary atypical pneumonia and 4·9 days in acute respiratory disease without pneumonia. Except for the pneumonic complication in the former these cases appeared to be similar. More than half the patients had a leucocytosis ranging from 11,400 to 16,300.

In retrospect this illness was thought to resemble the syndrome of undifferentiated acute respiratory disease (ARD) described by the Commission on Acute Respiratory Disease[5] and the syndrome of febrile catarrh, which was distinguished from influenza by Stuart Harris and his associates.[6]

Berge and his associates,[7] in a laboratory investigation of febrile acute respiratory infections occurring among military recruits at Ford Ord in California between December 1953 and July 1954, noted that less than 10% of the patients studied could be shown to have influenza. A similar proportion had disease of streptococcal origin or caused by viral agents other than influenza or the APC group. About 80% of the cases were diagnosed clinically as primary atypical pneumonia or other acute respiratory disease, including bronchitis, pharyngitis, common cold, tonsillitis, and upper respiratory infection. Less than half of the cases showed serological evidence of infection with APC agents. From throat washings of these patients, a total of 51 viral agents of the APC group were recovered in tissue culture. Among these were 3 immunological types: Type-3 14%, Type-4 43%, and Type-7 43%. Type-4 agents appeared to be associated to a large degree with infections involving the lower respiratory tract and Type-7 strains were most frequently found in affections of the upper respiratory tract.

The occurrence of viruses of this group has also been recently reported from England by Zaiman and his associates.[8]

The name 'Adenovirus' has recently been proposed for this newly discovered group of respiratory-tract viruses.[9]

RECENT EXPERIENCE IN SOUTH AFRICA

Several outbreaks of an illness similar to the infections described above were seen early in the 1939-1945 war amongst the recruits in military camps in various parts of the Union of South Africa. Studies of these outbreaks were undertaken by the staff of No. 2 Mobile Laboratory, South African Medical Corps, in the Zonderwater, Ladysmith and Piet Retief camps. These failed to reveal the influenza virus. The onset of the illness was not sudden as it is in influenza but was rather insidious. Most patients complained of sore throat and were found to have tonsillitis and pharyngitis with a mucopurulent exudate. In particular it was noted that many became hoarse and complained of a dry dusty irritating feeling in the throat associated with a spasmodic cough yielding a slight amount of mucopurulent sputum. Some developed pneumonic signs and symptoms and on clinical grounds were diagnosed as having primary atypical pneumonia. Most patients were feverish and the fever lasted from one day to a week, only occasionally longer in the absence of pneumonic involvement. In several camps meningococcal meningitis became epidemic during or after these outbreaks of respiratory infection and many of the patients developing meningitis gave a history of such respiratory infections immediately preceding the onset of the meningococcal infection. It was considered that this respiratory infection may have lowered the patients' resistance as well as providing more favourable conditions in the upper respiratory tract for the meningococcus to flourish. In this way the respiratory virus may have acted as a provoking factor in the development of meningitis.

Influenza virus was isolated in 1941 from cases of a typical outbreak of influenza occurring in Zonderwater camp, but not from these other outbreaks of respiratory disease.

Since that time several outbreaks of a similar disease have occurred in various parts of South Africa and attempts have been made to isolate influenza virus from cases. These have been unsuccessful. On the other hand

influenza virus has been readily isolated from a representative sample of cases in several typical epidemics of influenza. These tend to develop, reach a peak and subside much more rapidly than the other outbreaks of respiratory disease, which tend to smoulder for many weeks. Unlike typical outbreaks of influenza, which usually occur in the late winter, the outbreaks of ARD may occur in other seasons as well. In retrospect it seems certain that some at least of these outbreaks of non-influenzal respiratory disease were caused by this newly discovered APC group of viruses. It is therefore of interest to note that attempts to isolate virus from tonsillar tissue removed at operation at the Transvaal Memorial Hospital for Children have been successful in 8 instances. Two of these viruses have been identified as herpes virus. The other 6 have the characteristics of the APC group. The findings of the detailed study of these viruses will be reported when these have been completed. In the meantime their occurrence in South Africa is noted and the characteristics of the diseases they cause have been described. The steps to be taken to establish their diagnosis will also be of interest.

LABORATORY DIAGNOSIS

The diagnosis may be established by the isolation of the virus or by the demonstration of the development of specific antibodies during and after the infection.

Virus Isolation

As shown originally by Rowe and his associates, the virus may be isolated from tissue fragments of tonsils or adenoids. These tissues when planted under suitable conditions in test-tubes continue to grow, producing outgrowths of epithelial cells and fibroblasts. At varying intervals of time extending up to 72 days in some cultures, the cells degenerated and broke away from the original explant. These cytopathogenic changes were progressive until the entire culture was involved. Sub-inoculation of the nutrient fluid from these cultures into monolayer cultures of other types of cells caused a similar type of degeneration. It was concluded that a specific agent was responsible for the degenerative changes in the cells.

The frequent and ready isolation of virus from tonsils or adenoids suggests that these viruses may persist in these tissues for a long time after the acute phase of the illness is over. However, isolation from tonsils and adenoids obviously is not a method of isolation which can or will be followed in many cases during the acute phase of the illness. The routine procedure during this phase is to collect throat washings obtained by gargling with nutrient broth. After suitable treatment with antibiotics—by adding 100 units of penicillin and 100 μg of streptomycin per millilitre—these are inoculated on to tissue cultures in test-tubes of Hela cells or of monkey kidney cells, or other cultures of human or monkey cells which have been found to be susceptible. Cytopathogenic changes may not be seen until about the 28th day after inoculation, but usually they appear before the 12th day. Sub-inoculation into fresh tissue-cultures usually produces changes within 5 days. These cytopathogenic changes are similar in appearance to those originally seen in cultures of human adenoids and tonsils. During the process, complement-fixing antigens are produced in the nutrient fluid of the tissue cultures.

Complement-Fixation Tests

The complement-fixation test provides a simple and rapid method for confirmation of the presence of APC virus in tissue culture. The antigen is a non-infective soluble substance, which can readily be separated by differential centrifugation or by filtration, and has been shown by differential centrifugation and electron microscopic studies to be smaller than the infective particle. These studies have also shown that the latter are spherical particles ranging from 80 to 120 mμ.

Neutralization Tests

Neutralization tests with specific antisera prepared in rabbits have revealed that there are at least 14 antigenically distinct types of APC virus. When a virus has been isolated and its relationship to this group established by complement-fixation tests, it is typed by noting which specific antiserum neutralizes its cytopathogenic action.

Pathogenicity for Laboratory Animals

Unlike the influenza virus, which may be propagated in several animal hosts, the viruses of the APC group so far have proved non-pathogenic to laboratory animals or embryonated eggs. However, the isolation of these agents from the nasopharynx of monkeys has been reported. This lack of pathogenicity somewhat limits the investigation of the diseases produced by this group, but most aspects of such study can be covered adequately by the application of the tissue-culture technique.

Prophylactic Vaccination

This tissue-culture method may also be applied to the development of a prophylactic vaccine. The need for such a vaccine is particularly apparent in military camps of newly recruited soldiers. There would also be scope for its use in the civilian population. For example, its administration to the staffs of hospitals and similar institutions and of factories, in preventing the more serious results of infection, might considerably reduce absence caused by illness. It is therefore of interest to note that Hilleman and his colleagues[10] have reported the successful production of such a vaccine, containing 2 types of virus. It still has to be shown that vaccine can be produced on a large scale, though no difficulty should arise to prevent this. However, in view of the large number of types of virus involved, it may be difficult to include sufficient antigen of each type in an ordinary dose. Whether it will be possible and necessary to include all the types in a vaccine is a problem still to be studied. Future studies will also provide an answer to the question whether a vaccine will be effective in preventing the majority of cases.

SUMMARY

The characteristics of the newly discovered group of APC viruses are described. It is noted that they have been incriminated as an important cause of acute respiratory disease. The features of these infections are

described and it is noted that the signs and symptoms include sore throat, pharyngitis, laryngitis with hoarseness, cough yielding mucopurulent sputum, and fever. Some cases present with the features of primary atypical pneumonia.

The isolation of APC viruses in Johannesburg is recorded. The laboratory procedures to establish the diagnosis of infection by this group of viruses are briefly described.

The authors are grateful to Dr. H. H. Malherbe of this Institute, who arranged with the staff of the Ear, Nose and Throat Department of the Transvaal Memorial Hospital for Children to supply the tonsillar tissue used in their studies.

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CEREBRAL ANOXIA FOLLOWING ANAESTHESIA

WITH ILLUSTRATIVE CASE REPORT

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It is perhaps surprising that anoxic damage to the brain, which derives most of its energy from the oxidation of glucose, does not occur more often during inhalation anaesthesia. Anaesthesia itself produces a degree of histotoxic anoxia and if this degree is exceeded irreversible damage to nervous tissue may occur.

If cerebral damage does manifest itself after an anaesthetic it is often difficult to determine exactly what has gone wrong. Modern anaesthesia has become a complicated procedure that may involve the use of a number of different drugs and techniques. These include morphine or barbiturates (for pre-medication or induction), muscle relaxants, and the deliberate induction of hypotension, in addition to the main anaesthetic agent. These may all contribute either directly or indirectly to a tragic accident.

Morphine and the barbiturates not only depress the respiratory centre but are capable, if given in excess or to a susceptible subject, of causing cerebral damage in their own right. The coma of barbiturate overdosage is a familiar sight to many doctors and the fact that barbiturate coma is sometimes used instead of electroconvulsion in the treatment of the psychoses underlines their ability to cause profound cerebral changes.

Muscle relaxants have been hailed by surgeon and anaesthetist as almost indispensable in achieving a smooth and swift induction. Beecher and Todd[1] state that the number of anaesthetic deaths increases 6-fold with the use of relaxants. They suggest that in some cases death may result from acute circulatory collapse from the ganglionic blocking action of relaxants. Beecher and Todd dealt only with cases who died, but one may well feel disturbed by this factor of circulatory collapse in patients who survive an anoxic episode. Indeed many anaesthetists seem surprisingly casual about the danger of meddling with the circulatory systems of their patients. The deliberate induction of hypotension during surgery is often undertaken very lightheartedly. Many surgeons and anaesthetists fail to realize that a 'bloodless field', although a technical delight, is also an anoxic field. It is small comfort to relatives to know that a patient's operation was technically perfect if one of the side-effects of that operation is dementia from cerebral anoxia. It has been well recognized for years that sudden vascular collapse, e.g. after severe haemorrhage, coronary occlusion, spinal anaesthesia etc. can have profound cerebral effects. Cases of pituitary failure resulting from severe postpartum haemorrhage are a special group where the site of cerebral damage is determined by the puerperal state.

Bedford[2] states that 'hypotensive surgery is unjustifiable in people of any age-group and particularly in the elderly'. He claims that many old people are permanently disabled in a cerebral sense after operations because their pre-operative cerebro-vascular insufficiency left no safety margin for any extra anoxic stress.

In addition to these new hazards accompanying modern anaesthesia the old dangers still account for a fair number of cases of anoxia. Routine checking of anaesthetic equipment should never be a casual business —a faulty airway or an incorrect mixture may be the direct cause of grave neurological damage or death. The choice of the main anaesthetic agent also demands careful consideration. One does not need to stress the notorious reputation of nitrous oxide. Courville,[3] in 1939, had no difficulty in finding enough cases of anoxia from the use of this gas to write a book on the subject. It is now well recognized that nitrous oxide cannot be used for deep or prolonged anaesthesia without producing a dangerous degree of anoxia.

The clinical picture during and after anoxia varies. Failure of respiration or circulation or both usually

marks its onset but in some cases there is no indication that anything unusual has occurred until the patient fails to regain consciousness after withdrawal of the anaesthetic. This may lead to a surprising amount of confusion about what has actually happened to the patient. This is particularly true of neuro-surgery, where operative damage to the reticular substance of the brain stem may produce profound disturbance of consciousness.

Patients who die may do so within a matter of hours, may appear to recover and then suddenly relapse, or may linger with persistently disturbed consciousness for days, weeks or even months before dying. Courville[4] states that as a general rule if recovery is to be complete then all symptoms should disappear in 24 hours.

The state of emerging consciousness may be marked by motor restlessness, convulsions, abnormal behaviour etc.

If signs of cerebral damage persist these may take a variety of forms. A rough division into the following sub-groups is useful, though it should be remembered that practically any part of the brain may be damaged: (1) Organic dementia, (2) a parkinsonian clinical picture, (3) choreo-athetosis.

Organic dementia in the form of gross intellectual deterioration is fortunately not very common, but may occasionally be severe enough to necessitate the certification of a patient. Lesser degrees of intellectual impairment are commoner, e.g. forgetfulness, loss of the power of concentration etc. This intellectual impairment may well be linked with the personality changes so often seen after cerebral anoxia. These consist of irritability, lack of emotional control, and a tendency to withdrawal. Although this personality change is often a subtle one and not perceived by doctors, it can make a patient extraordinarily difficult to live with. Lucas[5] described a case where a patient with a morose and aggressive personality was greatly improved by an anoxic episode, becoming both placid and pleasant. This is strongly reminiscent of the change which may occur after a pre-frontal leucotomy.

A parkinsonian clinical picture has been recognized for years as a complication following exposure to carbon monoxide, or other anoxic stress, e.g. nitrous-oxide anaesthesia.

Choreo-athetosis as an isolated manifestation is not common. Courville[6] described a case of a woman of 19 who sustained anoxia during nitrous-oxide anaesthesia for appendicectomy. She was unconscious for 3 days, during which time several seizures occurred. She was blind for 3 months and unable to make herself understood until the 6th post-operative month. 'During this time she developed purposeless and uncontrolled movements of the extremities, which became so rigid and contracted that she was unable to get about.' Courville emphasizes the rarity of this type of clinical picture and says that no counterpart of this case was found in the available literature. He also points out that in patients who only survive for a few weeks or months this clinical picture is not seen, although at autopsy severe damage to the lenticular nucleus may be found.

In 1942 Kasin and Parker[7] reviewed the literature dealing with cases of choreo-athetosis following nitrous-oxide anaesthesia. They found only 6 cases in whom choreo-athetosis was mentioned and then always as one of many other clinical manifestations. They reported in detail on a case in whom choreo-athetosis appeared as the major symptom. Their case is almost identical with the case reported in this paper.

The clinical symptoms depend on the site and extent of cerebral damage. There are certain general principles underlying the pathology: That grey matter is more vulnerable than white matter; that the more vascular parts of the cortex, e.g. the visual and parietal areas suffer most; that certain of the cortical layers are more involved than others, so that focal and generally asymmetrical damage is a feature; that basal ganglia are extraordinarily susceptible to anoxia; and finally, interestingly enough, that the relatively avascular white matter may occasionally undergo progressive demyelination.

There are undoubted variations in the clinical picture depending on the anaesthetic agent used, e.g. the curious vulnerability of the visual area to nitrous oxide etc., but what precisely determines the variations in the clinical picture is not known. In the same way the pathogenesis of the symptoms and signs is often puzzling. Courville[8] invokes a vasomotor factor as well as the factor of direct anoxic damage to nerve cells. It may work through a general vasomotor instability of the cerebral circulation. This depends on damage to central vasomotor control, or else it acts more directly by anoxic damage to cerebral capillaries, causing a temporary dysfunction which may resolve completely or ultimately lead to endothelial proliferation resulting in ischaemic damage to nervous tissue. Courville's vascular theory is attractive in many ways. It explains the waxing and waning of the clinical signs and also the occasional inexorable progression of neural damage months or years after the original anoxic episode. It has also stimulated other workers to treat acute cerebral anoxia by reducing cerebral oedema.[9] Argent and Cope[10] state that sucrose (50%) is the ideal substance to use for the reduction of cerebral oedema. Unlike glucose it does not cause any 'rebound oedema', because of its strong diuretic quality. These authors feel that intravenous sucrose should be administered to any case of coma in whom cerebral hypoxia has been an etiological factor.

Despite enthusiastic reports of recovery from cerebral anoxia by reduction of cerebral oedema many patients remain permanently damaged. It is clear that great care on the part of the anaesthetist and surgeon is imperative to avoid such tragedies. One of the chief difficulties, as with much of the practice of modern medicine, is that the anaesthetist only sees the patient for a short time and for a subsidiary purpose. The evolution of an anoxic episode is seldom seen in its totality, except possibly by the general practitioner, so that the full impact of an anoxic tragedy is not experienced by those primarily responsible for it.

The following case illustrates many of the points that have been discussed.

CASE REPORT

The patient was a healthy male of 24. On 3 June 1955 he was operated on for acute appendicitis. Anaesthesia was induced with 0·9 g. of thiopentone sodium, to which 180 mg. of Gallamin had

been added to induce relaxation. A nasal endotracheal tube was passed and anaesthesia maintained by nitrous oxide and oxygen (25%) administered by intermittent positive pressure. During the operation the patient became intermittently cyanosed and after 30 minutes it was discovered that the tip of the endotracheal tube only passed the glottic opening when the patient's head was extended. If his head was flexed the tip of the tube fell short and its lumen became obstructed. (The condition of the tube was apparently not very satisfactory; otherwise it could not have been obstructed so easily.) The patient was immediately ventilated with an 80% oxygen mixture. All in all it was estimated that the duration of anoxia, which had been intermittent, was about 15-20 minutes.

After the operation the patient took some time to regain consciousness and when he did so he was restless, violent and confused. The surgeon saw him 8 hours after the operation and by this time he was correctly orientated and recognized the surgeon. During the first 30 hours after the operation he had 4 injections of ½ gr. of omnopon and about 15 c.c. of paraldehyde intramuscularly. As the effect of sedation wore off he would begin to grimace, chatter irrelevantly and throw his limbs about. He continued in this state with slight improvement until on the 9th post-operative day he suddenly became completely disorientated and surly and refused to speak. Omnopon, pethidine, largactil and paraldehyde did not improve his condition.

He was transferred to a private nursing home after some days and on arrival was so much improved that for 2 days he was walking around the grounds and, except for an occasional grimace, appeared completely normal. He then relapsed, becoming restless and unmanageable and assaulted one of the nurses without provocation. He was then transferred to Groote Schuur hospital 20 days after the operation.

On admission he was so restless and violent that it was impossible to examine him fully. He presented the picture of gross dystonia, with violent, unceasing movements of the face, trunk and upper limbs. He was, however, fully conscious and correctly orientated, which gave the whole clinical picture a bizarre and pathetic note. As the prognosis was thought to be extremely poor the main therapy was directed to securing some rest for the patient. The anaesthetic staff put up a pentothal drip and he received 12 g. of pentothal intravenously in the next 2½ days. While the drip was running the patient was quiet and still. When it was discontinued the involuntary movements recurred but were less violent. For the next 10 days he was given 18 gr. of sodium amytal and 300-400 mg. of largactil daily, with an occasional injection of 3 gr. of sodium gardenal.

At the end of 10 days he suddenly seemed much better. He was less restless and movements of his face, trunk and limbs were just perceptible. His main trouble at this stage was a large abscess in his buttock. This was incised under pentothal, nitrous oxide and oxygen and a large amount of pus was drained. The improvement in his clinical condition was sustained, though occasionally interrupted by episodes where he would shout for no reason, tear his dressing gown, or urinate on the floor.

Five weeks after admission he was able to hold his arms and hands outstretched without dystonia and his facial grimacing was just perceptible. Seven weeks after admission his wife (a good observer) thought that his condition was normal. She said that he had always had a tendency to blink and grimace when talking and that his somewhat fatuous behaviour was not abnormal. He was discharged after having been in the hospital almost 2 months.

Three months after his discharge his wife reported that he had just begun to work and had at the same time begun to experience 'giddy turns' during which he looked dazed and became unsteady on his feet. These episodes lasted a few seconds and occurred once or twice a week. They became progressively more frequent until 3 months later he was getting them 4-5 times a day and was forced to stop work because they were so incapacitating. In addition his limbs and face had begun to twich frequently and he had become so depressed that he contemplated suicide. The following month he had one generalized seizure and at this stage reported back to a neurologist. The attacks were considered epileptic and he was put on luminal, ½ gr. *tds*, and epanutin, 1½ gr. *bd*. At the end of 2 months of regular medication he was so much improved that he returned to work. His wife was very pleased at his improvement but remarked on the obstinacy of his behaviour since his illness. She said it was in marked contrast to his previous placidity, understanding and desire to cooperate with her. She also remarked on his newly acquired irritability and the tendency of his hands to twitch if he got excited. She did not think he showed any intellectual deterioration and his previous interest in sport etc. had remained unaltered.

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BILATERAL VARIATION BETWEEN EARS IN PITCH DISCRIMINATION*

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I

The physiological phenomenon to which I would draw attention and which apparently has never been systematically studied, was brought to my notice quite accidentally several years ago. I refer to the fact that, during the last war, I found that my two ears—the ears of a professional musician with a keen perception of musical pitch—heard any given sound with two different frequencies, when tested separately.

The way in which I discovered this fact was as follows: It was in 1946 in Johannesburg when I was needing two tuning-forks of the 'low' or 'New Philharmonic' pitch (in which C=522 vibrations per second). These I did not possess, though I had several of the 'high' or 'English' pitch (in which C=540 vibrations per second). But by filing a notch across the 'bow' of a tuning-fork, near the handle, one can cause the tines to lose some of their elasticity, and consequently lower the pitch of the fork. I accordingly borrowed a standard 'low-pitch' fork tuned to C=522 vibrations per second, and a small triangular file, and proceeded to lower the pitch of two 'high-pitch' forks. Being right-handed I laid the 'prototype' fork on the table to my right, and the two to be altered to my left. I then began to file a notch in one of the latter, checking the result by sounding it close to my left ear, while I sounded the 'prototype' near my right ear.

Now, as is well known, when two forks are in perfect unison, no beating (or throbbing) occurs between them when they are sounded together; but if their frequencies do not agree exactly, beats are heard. I filed each 'high-pitch' fork until when checked separately with the 'prototype' no beats were noticeable.

I then sounded the two re-tuned forks together, using the table as a resonator. Since no beats were audible, I rightly concluded that they had identical frequencies, and assumed that

* A paper read at the meeting of the South African Association for the Advancement of Science (joint meeting of Sections D and E), Grahamstown, July 1955.

these frequencies were identical with that of the 'prototype'. At this stage I sent all three forks to Professor Howard H. Paine, head of our Department of Physics at the Witwatersrand University, who very shortly telephoned to tell me that, although my two re-tuned forks were in perfect unison, each of them, when sounded with the 'prototype' on a common resonator, such as a table, produced beats.

There could be only one explanation of the phenomenon; my two ears interpreted the vibrations of identical forks differently. I therefore sounded the re-tuned forks together, first at my right ear, and then at my left; on neither occasion were beats audible. But, when I sounded the pair one at each ear, beats were heard. Further, I soon became conscious of the fact that the sound of a fork held near my right ear was very slightly, though appreciably, sharper than that of the same fork when held near my left ear.

At the time I described the phenomenon to several of my medical colleagues, who assured me that they had never heard of it. They, however, thought that it might in all likelihood be the normal state of affairs, since pairs of organs in the human body are seldom, if ever, exactly matched.

Apparently very little has been written on this subject. I found a brief reference to it in an issue of *The Musical Times* of the early twenties, and nothing further until I read recently of a London musician in whom the pitch discrepancy between his two ears was so marked that it was actually painful to him, and that he had been successfully operated upon to bring his ears into better accord. But no information was vouchsafed on how this was accomplished, or by whom. I therefore thought it advisable to put my own experience on record.

The ear specialist is primarily concerned with the general capacity of his patients to hear sounds of varying pitch, with the relative intensity of these sounds, and with their frequency range; but only secondarily with fineness of pitch discrimination, and apparently rarely, if at all, with differences between the ears in respect of such discrimination. But it would seem that here there is a fruitful field for both physiologist and psychologist. Is this phenomenon normal? Does it vary as between musician and layman? Or between youth and age, sex and sex, and race and race? It would appear that it would provide material for an interesting statistical dissertation, which should commend itself to a student on the look-out for a subject for a novel and worthwhile thesis.

II

Since I wrote this paper and have discussed it with colleagues, I have found that the phenomenon which I described has not only been known to physiologists, but has been discussed in a number of physiological papers. As the references with which I have now been made acquainted all appear in journals which I could hardly be expected to meet with, I make no apology for my ignorance of them, particularly in view of the fact that they seem to have been equally unknown to many professional men, including medical men and psychologists, to whom I mentioned the phenomenon. And, needless to say, no musician with whom I am acquainted had ever heard of their existence or of the phenomenon itself. It should, however, be noted that the phenomenon of 'beats' arising when sounds of identical pitch are heard simultaneously by the two ears, and which were experienced not only by myself but also by several colleagues, does not appear to have been studied before.

The first 4 references were sent to me by my friend and colleague Dr. Jaap Kunst of Amsterdam (the eminent Dutch musicologist), with whom I discussed my paper while I was in Holland a few weeks before our congress in Grahamstown. Although the phenomenon which I described was new to him he told me, after some thought, that he had some recollection of having seen several references to something like it which were published in Germany a good many years ago, and promised to look them out and send them to me. This he did, and I received them shortly after I had read my paper to our Association. They are as follows:

von Hornbostel, E. M. and Wertheimer, M. (1920): *Ueber die Wahrnehmung der Schallrichtung*. S.B. preuss. Akad. Wiss., Berlin, **20**, 388.

Halvorsen, H. M. (1920): *Binaural Localization of Tones as Depending upon Differences of Phase and Intensity*. Amer. J. Psychol., **33**, 178.

von Hornbostel, E. M. (1923): *Beobachten über ein- und zweiiriges Horen*. Psychol. Forsch., Berlin, **4**, 64.

Idem (1924): *The Psychophysiology of Monotic and Diotic Hearing*. Proc. and Papers, VII Int. Congr. Psychol. Oxford, 1923, p.377. Cambridge University Press.

Several weeks later I met Prof. D.W. Ewer, of the Department of Zoology at Rhodes University, who called my attention to an article by Robert Galambos (1955: Physiol. Rev., **34**, 499) entitled 'Neural Mechanisms of Audition', in which the phenomenon I have described, though without mention of 'beats', is referred to in a brief paragraph as a 'well-known *abnormality* of pitch experience, diplacusis' (my italics). According to this writer, 'the pitches perceived in each of two ears, interestingly enough, need not be the same. Normal subjects may report a significantly different pitch experience to the same tone *if this is delivered to first one, then the other ear*' (my italics again). In support of this statement Galambos quotes the following 3 references:

Jeffress, L. A. (1949): Amer. J. Psychol., **62**, 1.

Stevens, S. S. and Egan, J. P. (1941): Psychol. Bull., **38**, 548.

Wever, E. G. (1949): *Theory of Hearing*, New York: John Wiley and Sons.

Two other references deal with the occurrence of the phenomenon in subjects whose hearing is defective in various ways:

Davis, H. *et al.* (1939): *J. Neurophysiol.*, **2**, 500.

Rüedi, L. and Furrer, W. (1946): *Schweiz. med. Wschr.*, **76**, 843.

Finally, Galambos suggests that 'the available facts make it likely that some deficiency in neural inflow from one cochlea is the fundamental explanation for diplacusis'.

It will be noticed that these modern references all deal with the phenomenon primarily from the point of view of the physiologist and, as I have said, *the phenomenon of 'beats' arising between the ears appears to have been overlooked*.

None of these studies would seem to invalidate my appeal for a statistical study of the phenomenon; and in any event I think that the experience of a professional musician in this connection ought to be put on record.

'WHEN IN TROUBLE GO TO ETHER'

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Pretoria

My practice of using a 6-litre flow of oxygen per minute and no N_2O for prolonged anaesthesia has been looked on askance by my colleagues in Pretoria for many years now.

Is my success in some 20,000 cases in which a flow of only 6 litres of oxygen per minute was used perhaps due to the fact that in Pretoria we are at an altitude of 4,500 feet and under an atmospheric pressure of only 650 mm. Hg, whereas our instructions come from near sea level with a pressure of 760? Will CO_2 not diffuse more easily out of venous blood here than at the coast? It is generally accepted that it is more difficult to maintain anaesthesia here with gas than at the coast because it is more difficult to get enough N_2O into the blood. For the same reason it should be easier to diffuse CO_2 out.

It is said that my patients are in danger of (*a*) oxygen intoxication, and (*b*) anoxia, in spite of the use of pure oxygen as a carrier.

Oxygen intoxication at this altitude is impossible. It only occurs when oxygen is administered under a pressure of about 3 atmospheres. Anoxic anoxia is impossible with a flow of 6 litres and a

normal tidal volume. What you get with an insufficient tidal volume is deepening of anaesthesia owing to a chemical anoxia, even with an excess of oxygen and a pink patient. You are, however, more likely to get this with the same insufficient tidal volume when using gas and oxygen as a carrier. Here the anaesthetic action of CO_2 comes into play, due to the same chemical anoxia as with chloroform, ether etc.

CO_2 has 3 actions—or rather its action can be divided into 3 stages: (*a*) A stimulating action on the respiratory centre in low concentrations, (*b*) an anaesthetic action like that of other inhalant anaesthetics coming into play in concentrations of 10-40% and leading, if persisted in or if the CO_2 concentration exceeds this figure, to (*c*) a paralysing action on the breathing centre—and eventually the heart. CO_2 can even be safely used as an anaesthetic agent for short operations by the use of 30-40% of CO_2 in oxygen, provided the patient is first returned to pure oxygen and only later to normal air—i.e. provided he is first 'decarbonized'. It was used for this purpose 100 years ago.

I maintain that with a flow of 6 litres of oxygen neither excessive stimulation nor excessive anaesthetic action (chemical anoxia) leading to exhaustion or paralysis can occur as the result of CO_2 retention—provided of course there is no obstruction and no respiratory depression; in other words, provided a normal tidal volume is maintained. Where there is unavoidable partial obstruction it is still the best technique. Under such circumstances N_2O+O_2 as ether carrier would be positively dangerous.

Dr. Samson[1] in his excellent article in this *Journal* (19 May 1956) states that in the old open-ether technique too much CO_2 is blown off into the air, to the detriment of the concentration required to stimulate breathing. I do not agree. I maintain that there is at least 200 c.c. of expired air under the mask and in the bronchi for re-breathing (it is generally conceded that there is a deficiency of oxygen under the open-ether mask). The fresh air slightly charged with CO_2 inhaled through the mask, added to this 200 c.c. of retained air, is quite sufficient as a normal breathing-stimulant. Above all, ether itself is the only anaesthetic agent that in itself is a respiratory stimulant in moderate doses[2]—hence its safety. Dr. Samson also omits to mention a most important cause of anoxia, viz. an insufficient flow of oxygen; perhaps this is too obvious to mention, yet it is a frequent cause of anoxia. Nor do I follow his reasoning under the heading 'Maintenance of the Tidal Volume'; with a deficient tidal volume there is no 'super-saturation' with oxygen anywhere in the lung, as far as I am aware.

To give CO_2 in excess during gas and oxygen anaesthesia (especially when there may already be a deficiency of oxygen) is extremely dangerous. It is permissible only for very short intervals when the oxygen supply is sufficient. Even here, when indulged in for any length of time, the patient, after a long operation, though perfectly pink and breathing normally, may lapse into respiratory depression and anoxia or post-operative collapse when returned suddenly to normal air. Partial exhaustion or depression of the breathing centre takes place owing to the continued excess of CO_2 administered. This only comes to light when this excessive stimulation is withdrawn and the patient is returned to normal air. Patients should therefore always be put on pure oxygen before leaving the theatre.

A factor in the development of this post-operative collapse in long operations in which N_2O in high concentrations is used is the fact that nitrogen can displace 30 times its own volume of N_2O from the blood into the alveoli when the patient is suddenly returned to air. This sudden release of N_2O into the alveoli will obstruct the inward flow of sufficient oxygen and cause symptoms of anoxia and collapse. This mechanism probably also plays a part in the so-called cyclopropane collapse. During the first minute of N_2O anaesthesia no less than 1,000 c.c. of N_2O is absorbed by the blood. In 1-3 hours from 7 to 30 litres can be absorbed.[3] Saturation is only reached after about 5 hours.

It may be held that I make the technique of my speciality too simple in using ether and oxygen in all but the short cases and those with an explosion hazard. I reply that the safety of the patient is the most important factor to consider, not the anaesthetist, the high-sounding technique or the surgeon. I maintain that the tendency nowadays is to use techniques that are much too complicated and dangerous, and are difficult to master without any compensatory benefit. It is already evident that cyclopropane is rapidly falling out of fashion. We try all sorts of new techniques and new drugs because they are new; they are often dangerous in our hands because they are new; and they are often found wanting when they are no longer new.

The ideal is to use no new drugs and techniques except in teaching hospitals, for all drugs and techniques are naturally dangerous in the hands of beginners. Here, whatever is taught should be supervised by teachers in whose hands, by the nature of their training, all techniques and drugs should be as safe as humanly possible. But even here we have to reckon with the human element, which is never infallible. To my way of thinking it is this personal element that is to blame for most of our anaesthetic catastrophes or near-catastrophes, and not so much the disease, the patient, the technique or the drug as such. This aspect of our practice seldom, if ever, comes to light at inquests.

In anaesthetic literature and teaching much stress is laid on the dangers of CO_2 *per se*. I think more stress should be laid on the necessity of giving an abundance (or superabundance) of oxygen. Draper *et al.* proved that in dogs a concentration of 63% of CO_2 in the alveoli was not inconsistent with life after 45 minutes of non-breathing in an atmosphere of oxygen! We should only avoid this building up of CO_2. The best way of doing this is to give oxygen pure with ether in prolonged cases. Life in Draper's dogs was maintained by what he calls 'diffusion respiration' considerably assisted in my opinion by the cardiac impure respiratory wave. Anaesthesia was maintained by CO_2.

Quite recently I was horrified to see a GP anaesthetist giving a flow of only 2,000 c.c. of oxygen per minute with ether, without gas. This necessitated the frequent lifting of the mask from the patient's face to admit air! Machines measuring a maximum of only 2,000 c.c. per minute should not be allowed in any hospital where persons other than specialists administer anaesthetics.

Of all anaesthetics ether is the only one which stimulates breathing—both in frequency and depth. It does so mainly by peripheral stimulation of the stretch and deflation receptors in the lung, and also by release of epinephrine and stimulation of other systemic receptors as well as the cerebral breathing-centre. Above all, of all anaesthetics, gaseous as well as intravenous, ether causes the least CO_2 retention in the system—the great danger with cyclopropane.

Recently Miller[4] found that with the usual anaesthetic techniques CO_2 is retained in the system more than we realize, to the detriment of the patient. The reason? Too little oxygen supplied. In my opinion, excessive post-operative vomiting is mainly caused by hypercarbia and anoxia—chemical anoxia as well as anoxic anoxia. Where these do not occur, post-operative vomiting is negligible and can be completely avoided by the pre-operative administration of Largactil.[5] This new introduction appears to depress the vomiting centre. It also potentiates relaxants, so that less anaesthetic and relaxant are required to maintain the patient in a satisfactorily relaxed state for surgery. (I was greatly concerned when one of my first patients on Largactil slept for 6 hours—not knowing that this was an occasional harmless side-effect.)

Anoxia and CO_2 retention can only occur from insufficient intake of oxygen and insufficient blow-off of CO_2 (i.e. insufficient tidal volume) or from depression of respiration by excess of ether and excessive premedication with narcotics.

In connection with tidal volume and CO_2 retention the size of the bag is of some importance too. Bags are usually of 1 or 2 gallon size. Personally I think only the smaller bag should be used—or better still one of half a gallon—or even quart size! No matter how well the blow-off valve is acting some exhaled gases will pass it; and more will go to the big bag than to the smaller ones because there is a little less resistance in the former—when the bag is being worked at near-full. This means that with the big bags more re-breathing takes place, which under certain circumstances may become excessive. Moreover when, in an emergency, pure oxygen is required it takes longer to empty the big bag of whatever it may contain than the smaller one.

CONCLUSION

In conclusion may I again stress that there are only 3 fundamental factors that have to be reckoned with in the administration of ether, viz. (*a*) a sufficient supply of oxygen, (*b*) a sufficient blow-off of CO_2, and (*c*) a sufficient concentration of ether (4-5%). (*a*) and (*b*) can be satisfactorily attained by a minute flow of 6 litres and a well-acting valve—in infants by a free passage-out with no valve at all (e.g. the Ayres tube)—and (*c*) by experience.

The old saying, 'When in trouble go to ether' still holds good.

I should however prefer to make it read, 'When in trouble go to oxygen and ether', or rather, 'Before you are in trouble go to oxygen and ether'.

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OPHTHALMOLOGICAL AND OPTOMETRICAL SERVICES IN THE UNION

A REPLY TO AN ARTICLE BY DR LUCAS YOUNG[1]

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The South African public is served by nearly 100 specialist ophthalmologists, registered as such by the South African Medical Council (77 in actual practice according to Dr. Lucas Young), and by about 280 optometrists (nearly all members of the South African Optical Association) whose qualifications are acceptable for registration by the Medical Council. Then there are 60 or so practising optometrists whose qualifications are not at present recognized, but who have had some training and might be expected to have a chance of passing a qualifying examination if one were set up.

Beyond these, there is a nebulous fringe of part-timers and chancers who, in the absence of legislation in the Union, are taking the opportunity of battening on the credulity of the public. Legislation when it comes, if it is to be effective in protecting the public, must exclude these.

The title of Dr. Young's article, 'A Public Ophthalmological Service', is somewhat misleading, for his main discussion centres around refraction and not ophthalmology. It is chiefly concerned with the fact that a portion of an ophthalmologist's work, namely refraction, is also performed by optometrists.

Comparison with Great Britain. Dr. Young estimates that in Great Britain there is one ophthalmologist to 20,000 of the population, and in the Union one to 30,000 of the *European* population. Of optometrists (sight-testing opticians) there are in Great Britain one to 7,000, and in the Union one to 9,000 of the *European* population. In the Union there is also the Coloured and Native population, which is a considerable factor in many practices. The non-Europeans' demand for eye services is rising steeply as their standard of living and education rises.

'*Not particularly appertaining*'

It has been claimed that refraction is an exclusively medical act. This view has not been accepted in optometry legislation in other countries, and in our country it has been laid down (Rex *v.* Saks) that 'testing eyes for errors of refraction is not an act particularly appertaining to the calling of a doctor'. History, too, is against any such assumption. Up to the time of Donders, in the middle of the last century, ophthalmologists were barely interested in refraction, and right to the end of the century at Moorfields, the famous London eye hospital, it was customary to send patients out to be tested by opticians when glasses were necessary.

Since the beginning of this century, there has been increasing participation by ophthalmologists in refraction. In England 20-25% of cases are refracted by doctors and 75-80% by optometrists. The proportion is probably not very different in South Africa.

The work of many ophthalmologists consists to a considerable extent in refraction. Dr. Young has gone on record, elsewhere, with the sentiment that ophthalmologists operate for *kudos* but refract to live. Earlier ophthalmologists made their living through medical and surgical treatment and, while no optometrist would begrudge a modern ophthalmologist those patients who are willing to pay a higher fee for refraction, this fact should not be forgotten.

A reply to specific points

The following comments on Dr. Young's article are submitted, and alternative proposals will be given at the end.

The estimate that 90% of non-Europeans under presbyopic age who wear glasses do so purely for show is very wide of the mark. In my experience at the Cape the proportion is nearer 10% than 90%.

His conclusion that there are not enough ophthalmologists in the country to take over the refraction of the entire ametropic public is putting it mildly. In Cape Town, for example, where there are 12-14 ophthalmologists, an appointment can seldom be booked in under a week, often only within 6 weeks (although urgent cases, such as acute glaucomas, ulcers etc., can be fitted in at shorter notice, provided they are backed by a persistent optometrist or doctor). What would happen if the ophthalmologists had the total 100% of refractions to attend to, instead of only 25% as at present?

With regard to the further statement, that 'any great increase in the number of ophthalmologists is likely to be a slow and dubious quantity', one asks why ophthalmology seems to have difficulty in attracting students. The main reason is the policy that has been adopted by the Ophthalmological Society itself—namely that ophthalmologists should go all out to get refractions. The result is that the prospective student finds that ophthalmology has become a comparatively 'bloodless' speciality, and he opts for something more exciting. If ophthalmologists performed all the refractions, in most of their cases there would not be even a drop of boracic, let alone blood. Would this make the speciality more attractive?

Why should cooperation and give-and-take between ophthalmologists and 'opticians' be too Utopian to happen? (Incidentally too, why the use of the word 'optician' instead of 'optometrist'? It was to meet the views of the Ophthalmological Society, who about 1934 objected to the term optician, but stated that they had no objection to 'optometrist', that we went out of our way to adopt it.)

A scheme whereby Provincial hospitals could be used to provide additional eye-clinics has much to commend it, but Dr. Young's suggestions are none too happy. It would have to be run largely by part-time ophthalmologists, but part-time—or more correctly side-line—'ophthalmologists' are not likely to be very efficient. It is not likely that a postgraduate course of 6 months will produce a good refractionist. 'Deep knowledge' of the medical side, as Dr. Young says, would also be out of the question; the more likely result would be an inefficient dilutee. Then the phrase 'refraction *en masse*' has a very sinster sound. Such a thing may suit some countries, but South Africans are accustomed to private consultations at which they can expect to have reasonable time and care devoted to their *individual* case.

Clinics may be a necessity for the low-income groups, but it is difficult to imagine from any clinic, however well run, that 'as time passes . . . an increasing number of the public will fall into the habit of attending the clinics rather than the optician'. Every example quotable points to the opposite. For instance, the Gluckman Report advised health centres all over the country to replace private practice, but it was under no illusions that clinics,

even if called 'centres', would be popular. The public was to be enticed with free medical service, and various forms of coercion were to be used to force doctors out of private practice. The Government has never seen fit to apply such coercion, and instead of hundreds of health centres, each with a staff of 50 or so—doctors, dentists, nurses, health visitors, chemists etc.—there are just a very few pilot centres, carrying on an excellent work, but catering only for the very poorest in out-of-the-way corners of the country.

The British National Health Service was to consist entirely of a hospital and clinic service. Till this could be provided, a 'supplementary' service through private practitioners, doctors, dentists, optometrists etc. was instituted as a stop-gap. As it has turned out, the clinic service never caught on, and now a commission of enquiry admits frankly that, compared with private arrangements, it would be costly, cumbersome, and unpopular.

Dr. Young's idea that at the clinic the ophthalmologist in charge must be the first to see each patient attending and the last to see him as he leaves and, in pathological cases, the one to attend to him in between, would be regarded as fantastic. Yet ophthalmologists' thinking, in any case involving the possible use of optometrists as refractionists, is apt to demand that a medical man must see the patient first and last, and in many cases first, last and centre, and this is so whether in private or in clinic practice. The whole trouble about the question of 'direct access' in the Supplementary Health Services Bill came about because it was suggested that anyone wishing to consult an optometrist about glasses should be forced to do so through a general practitioner. Dr. Young was not in favour of such a scheme, but his own is no better. Because the ophthalmologist would be the senior partner in any clinic scheme, there is no reason to suggest that he should do *all* the work.

Another scheme discussed by Dr. Young, is the one run by the National Ophthalmic Treatment Board (NOTB) in England. In this scheme, ophthalmologists and near-ophthalmologists work in business relationship with dispensing opticians to run a kind of practice intermediate between clinic and private practice. When the NOTB was started many years ago, it was financed from medical sources and staffed with difficulty. It has probably just paid its way, with financial assistance from time to time, but has never met a public need that was not being better met in other ways, and has never been popular with the public.

As is often the case with ophthalmologists, Dr. Young purports to see special problems and disadvantages in 'opticians' being allowed to attend to the needs of the public, and the following phrases crop up, 'undesirable features . . . in the service . . . offered by opticians', 'the problem of the sight-testing optician', 'if the relative problems persisted'. Let it be said at once that these 'undesirable features' and 'problems' do not exist in the public's mind nor in the optometrist's. They exist only in the minds of certain ophthalmologists.

The suggestion that 'no person who is financially interested in the supply of spectacles should be the final arbiter in a decision to prescribe them', and that the public will be persuaded to buy more pairs of spectacles than are necessary, is a greatly resented reflection on the profession of optometry. Optometrists are not drawn from some substratum of society, and are just as punctilious in giving professional advice as a dentist advising a denture, or a surgeon advising an operation, or a doctor advising an expensive course of treatment.

The weakness of Dr. Young's suggestions is that unless suitable optometry legislation is brought in, for which the South African Optical Association has long been campaigning, there will continue to be no protection for the public from the unqualified quack. The idea that a clinic scheme can be made so attractive that it will draw away optometrists' clients to the stage where the last few remaining optometrists can safely be legislated out of existence, is futile. *Three-quarters or more of the public will continue to consult optometrists and the duty of the legislature is to see that these optometrists are properly qualified. This means statutory registration and control.*

ALTERNATIVE PROPOSALS

Dr. Young has stated his plan, of which the foregoing is a criticism. His maps show clearly the limitations of ophthalmological practice and the considerably better coverage provided by optometrists, and the even better coverage provided by the hospitals. If there is to be a change, let it be for the better. Why not examine the position from the angle of improving present services, rather than going against the grain, trying to bring in something that would rile the public, break the optometrists, and frustrate the ophthalmologists?

Under present economic conditions, there are two types of services to be rendered: (1) A clinic service for those able to afford little or nothing, and (2) a private-practice service for those in better circumstances. It does not follow that both will always be necessary, but for the moment, let us assume that both should be improved by every means possible.

Improving clinic practice

At the Cape Town hospitals, as far as I am aware, no optometrist is at present employed either part-time or full-time. The ophthalmologists are often barely able to cope with the patients attending, and spend too much of their valuable time dealing with refractions. A few optometrists on the staff would ease the burden very considerably. Certain conditions would have to be laid down to give the greatest effectiveness:

1. There should be no regulation whereby an ophthalmologist *must* be the first to see a patient, except for obviously pathological cases. With refraction cases it would cause a bottle-neck and interfere with the smooth flow of patients. If a case seen first by the optometrist turned out to be pathological, he would naturally refer it.

2. It might be more convenient to allow the optometrist to interview and screen all cases, whether pathological or not. His basic knowledge would be such that, with a little specialized instruction in what the ophthalmologist required, he would speedly prove superior to the nursing staff.

3. The optometrist could do certain work on pathological cases delegated to him by the ophthalmologist, such as refraction, fields, colour vision etc, and orthoptic training if no orthoptist were available.

4. He could assist in keeping case records.

The effective use of optometrists would depend on 3 things:

(*a*) The optometrist should be allowed to improve the apparatus used for refraction; in most hospitals that the writer has seen or heard of, this is inadequate for quick and accurate work.

(*b*) An optometrist, on qualifying, should spend a year or so working in the eye department of a general hospital.

(*c*) There should be progressive salaries for those optometrists who wish to make the hospital service a career.

In remote hospitals unable to support an ophthalmologist, a great deal more could be accomplished by means of a team composed of general practitioners and optometrists, than is possible with G.P.s alone.

Improving private practice

In private practice, distribution needs improving both as regards ophthalmologists and optometrists. If legislation were brought in whereby the unqualified optometrist in the towns and the travelling quack in the country were put out of practice, an increase in the number of qualified optometrists would be possible. They would recognize and refer pathological cases, and ophthalmologists would receive an increasing *number* of such cases, and at increasingly *early stages* when success is more likely. Thus there would be greater saving of sight. Also with a higher proportion of therapy and surgery in ophthalmological practice, the speciality would attract more entrants. There are a dozen or more towns in the Cape Province at least, that could support an optometrist if travelling quacks were done away with, and 3 or 4 of the larger ones that could support an ophthalmologist.

A great deal more could be done if optometrists and ophthalmologists worked in close confidence with each other, than at enmity. One would like to make the suggestion that the present is a suitable time for discussion to take place between the Ophthalmological Society and the South African Optical Association in an endeavour to end their differences and devote their combined efforts to the good of the public.

1. Young, L. (1956): S. Afr. Med. J., **30**, 704.

SOUTH AFRICAN MEDICAL CONGRESS, DURBAN 1957

The 41st South African Medical Congress (and 20th Annual Scientific Meeting) of the Medical Association of South Africa will be held in Durban, Natal, on 16-21 September 1957. The headquarters will be at Red Cross House, Old Fort Road, Durban. The Congress office at present is at 112 Medical Centre, Field Street, Durban. Telegrams 'Medcongress'.

The Local Organizing Committee is as follows: *Chairman*, Dr. A. Broomberg; *Vice-chairman*, Dr. N. A. Rossiter; *Hon. Organizing Secretaries*, Drs. B. Crowhurst Archer and S. Disler; *Hon. Medical Secretaries*, Drs. J. Kelman Drummond and A. J. Wilmot; *Hon. Treasurers*, Mr. R. Gowans and Dr. F. Walt; *Members*, Drs. E. W. S. Deale, K. W. Dyer, R. Elsdon-Dew, G. D. English, H. Grant-Whyte, B. Morris, R. Mundy, N. R. Pooler, J. Stolp, Alan Taylor and J. C. Thomas, Prof. I. Gordon, and Messrs. R. C. J. Hill, C. J. Kaplan, L. Knox and A. G. Sweet-apple.

Sections. The scientific proceedings will be divided into the following 19 sections: Anaesthetics; chest diseases; dermatology; general practice; hospital administration; industrial and military medicine; medicine; neurology, psychiatry and neuro-surgery; obstetrics and gynaecology; ophthalmology; orthopaedics; oto-rhinola-yngology; paediatrics; pathology; physical medicine; public health; radiology; surgery; urology.

Plenary Sessions. At least two plenary sessions will be held, of which the subjects will be (1) 'The Prevalent Parasitic Diseases of Man in Africa', and (2) 'The Surgery of Repair'.

Sub-plenary Sessions. Three combined meetings of sections will be held under this name.

Sectional Meetings. Besides separate sectional meetings it is intended to hold as many other combined meetings as possible. For the discussion of subjects of common interest secretaries of various sections are urged to establish contact so that combined meetings may be arranged. Facilities will also be provided for Groups to hold their Annual General Meetings.

Submission of Papers

Members wishing to present papers at the Congress are asked to communicate with the Hon. Medical Secretaries as soon as possible. Attention is called to the following requirements of the Congress regulations:

For the plenary sessions papers shall not occupy more than 30 minutes in reading, and discussion shall be limited to 10 minutes for each speaker; the corresponding times for sectional meetings are 20 minutes and 7 minutes.

Both for plenary sessions and sectional meetings a typed synopsis not exceeding 500 words shall be submitted to the Local Organizing Committee not less than 90 days before the Meeting, and *a typed copy of the whole paper in duplicate*, not less than 60 days before the Meeting. Papers not submitted in time shall not appear on the agenda of the Meeting.

It shall lie with the Organizing Committee whether any of the papers accepted shall be printed in galley proof and distributed at the Meeting at the time of registration. If that is done, whether for plenary sessions or sectional meetings, the writer of the paper shall not read it in full, but will be allowed 15 minutes to give a summary or deal with special points.

Accommodation, Travelling Facilities and Concessions

Arrangements are in the hands of the S.A. Railways Travel Bureau, which is in a position to undertake inclusive booking for visitors requiring transport, hotel accommodation, etc. The Durban office of the Bureau is at Church Street (next to St. Paul's Church), Durban. Enquiries on these matters may be directed to Dr. B. Morris, 917 Colonial Mutual Buildings, West Street, Durban, who is Hon. Secretary of the Accommodation Committee.

Exhibitions

The *Trades Exhibition* will, as usual, constitute a central feature of the Congress.

A Scientific Exhibition will also be held and is expected to be of exceptional interest.

Doctors' Hobbies. The arrangements for this exhibition are in the hands of Dr. Morris Cohen, 82 Medical Centre, Field Street, Durban, with whom intending exhibitors are requested to communicate.

BOOKS RECEIVED RECENTLY IN THE WITWATERSRAND MEDICAL LIBRARY

Adams, J. C. Outline of orthopaedics. Edinburgh. Livingstone. 1956.

Bocock, E. J. Applied anatomy for nurses. Edinburgh. Livingstone. 1954.

Bourne, G. Biochemistry and physiology of bone. New York. Academic press. 1956.

Boyd, G. A. Autoradiography in biology and medicine. New York. Academic press. 1955.

Brooks, C. McC. Excitability of the heart. New York. Grune & Stratton. 1955.

Dispensatory of the United States of America. 25. ed. Philadelphia. Lippincott. 1955.

Ecker, A. Normal cerebral angiogram. Springfield. Thomas. 1951.

Ellis, R. W. B. Child health and development. 2. ed. London. Churchill. 1956.

Engle, E. T. Hormones and the aging process. New York. Academic press. 1956.

Gaebler, O. H. Enzymes. New York. Academic press. 1956.

Gardner, L. Adrenal function in infants and children. New York. Grune & Stratton. 1956.

Glees, P. Neuroglia: morpyology and function. Oxford. Blackwell. 1955.

Great Britain. Ministry of health. Report of the committee of enquiry into the cost of the National Health Service. London. H.M.S.O. 1956.

Greenfield, J. G. Spino-cerebellar degenerations. Oxford. Blackwell. 1954.

Gustavson, K. H. Chemistry and reactivity of collagen. New York. Academic press. 1956.

Heilbrunn, L. V. Dynamics of living protoplasm. New York. Academic press. 1956.

Hofer, H. Primatologia. Basel. Karger. 1956. v. 1.

Hyman, H. T. Handbook of differential diagnosis. Philadelphia. Lippincott. 1953 repr. 1955.

International Congress of Biochemistry. Proceedings of the third international congress. Brussels. 1955. New York. Academic press. 1956.

Kjellberg, S. R. Diagnosis of congenital heart disease. Chicago. Yearbook publishers. 1955.

Lancet. Management of burns. London. Lancet. 1954.

Lyon, E. Virus diseases and the cardiovascular system. New York. Grune & Stratton. 1956.

Manson-Bahr, Sir P. History of the School of Tropical Medicine in London. London. Lewis. 1956.

Marinacci, A. A. Clinical electromyography. Los Angeles. San Lucas press. 1955.

Merrill, J. P. Treatment of renal failure. New York. Grune & Stratton. 1955.

Michels, N. A. Blood supply and anatomy of the upper abdominal organs. London. Pitman. 1955.

Moseley, H. F. Textbook of surgery. 2. ed. London. Kimpton. 1955.

Oster, G. Physical techniques in biological research. New York. Academic press. 1956. v. 3.

Paterson, D. Modern methods of feeding in infancy and childhood. 10. ed. London. Constable. 1955.

Patte, E. Les Neanderthaliens, anatomie, physiologie, comparaisons. Paris. Masson. 1955.

Pearce, E. C. Anatomy and physiology for nurses. 13. ed. London. Faber. 1956.

Perrodin, C. M. Supervision of nursing service personnel. New York. Macmillan. 1954.

Pincus, G. The hormones. New York. Academic press. v. 3. 1955.
Reynolds, R. J. S. Physical measures in the treatment of poliomyelitis. London. Faber. 1956.
Roberts, F. Medical terms. 2. ed. London. Heinemann. 1956.
Russell, R. J. Culture worlds. New York. Macmillan. 1954 reprinted.
Samuels, S. S. Diagnosis and treatment of vascular disorders. Baltimore. Williams & Wilkins. 1956.
Schamroth, L. Introduction to electrocardiography. Cape Town. Juta. 1956.
Symington, J. Atlas illustrating the topographical anatomy of the head, neck and trunk. Edinburgh. Oliver & Boyd. repr. 1956.
Underwood, E. J. Trace elements in human and animal nutrition. New York. Academic press. 1956.

PASSING EVENTS : IN DIE VERBYGAAN

Mr. I. M. Meresky, F.R.C.S., has commenced practice as a specialist surgeon at 212-213 Medical Centre, Welkom, O.F.S. The telephone number for messages will be 351.

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World Medical Association. The Second Secretariat Liaison Conference is being held in New York on 19/20 October 1956, for the purpose of providing an opportunity for Secretaries of Member Associations, the Editors of their Journals, and Members of Council to meet at the WMA Secretariat to discuss common problems. Thirty representatives from 15 Member Associations are expected to attend. The American Medical Association is to entertain the Conference to a dinner party on 19 October.

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Research Forum, University of Cape Town. The next meeting of Research Forum will be held on Tuesday, 6 November 1956 in the A-Floor Lecture Theatre, Groote Schuur Hospital, Cape Town, at 12 noon. Speaker: Dr. J. B. Herman. Subject: Gout and Diabetes Mellitus.

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Dr. J. Fine, ear, nose and throat surgeon, after mutual dissolution of partnership, is practising at 706 Medical Centre, Jeppe Street, Johannesburg. Telephones: 22-1402 and 22-1832.

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The Second World Conference on Medical Education will be held in Chicago, USA, from 30 August to 4 September 1959. The subject will be the continuing education of the doctor after graduation from undergraduate medical schools (Medicine a Life-long Study). The Conference will comprise a plenary session at which the First Conference on Medical Education (London, 1953) will be reviewed. This will be followed by meetings of 4 sections, namely, Section I—Basic Clinical Training for all Doctors; Section II—Advanced Clinical Training for General and Specialty Practice; Section III—Training for Research and Teaching; Section IV—Continuation Medical Education. The Conference languages will be English, French and Spanish, but German may be added. This Conference will be convened under the auspices of the World Medical Association in collaboration with the World Health Organization and the International Association of Universities. National Medical Associations and Medical Schools of the World are invited to assist in planning, recommending topics and speakers, sending representatives and providing publicity.

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Union Department of Health Bulletin. Report for the 7 days ended 27 September 1956.

Plague: One (1) case. Coloured female in the Kenhardt district. Diagnosis confirmed by laboratory tests.

Smallpox: Nil.

Typhus Fever: One (1) case. Native female in the Cradock district. Diagnosis based on clinical grounds only.

Epidemic Diseases in Other Countries.

Plague: Nil.

Cholera in Calcutta (India), Chalna (Pakistan).

Smallpox in Calcutta, Cuddalore, Karikal, Madras, Pondicherry, Tuticorin, Visakhapatnam (India), Dar es Salaam (Tanganyika).

Typhus Fever: Nil.

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Service for Doctors. On Sunday evening 14 October divine service was held at St. Stephen's Church, Pinelands, Cape, to which doctors were specially invited. The service was conducted by the Rector, Rev. L. A. Davis, who also preached, and the lessons were read by Drs. H. G. O. Owen-Smith, president of the Cape Western Branch of the Medical Association of South Africa, and Dr. A. W. S. Sichel, chairman of Federal Council. A large congregation attended.

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South African Paediatric Association. The next meeting of the Cape Town sub-group of this Association, will be held on Friday 2 November 1956 at 8.15 p.m. in the E-Floor Lecture Theatre, Groote Schuur Hospital, Cape Town. Dr. W. Emdin will speak on (*a*) The VIII International Congress of Paediatrics held in Copenhagen in July 1956 and (*b*) his trip beyond the Arctic Circle, to Lapland and North Cape.

REVIEWS OF BOOKS : BOEKRESENSIES

HEART DISEASE

Treatment of Heart Disease. A Clinical Physiologic Approach. By Harry Gross, M.D., F.A.C.P. and Abraham Jezer, M.D. Pp. x + 549. 91 Figures. $13.00. Philadelphia and London: W. B. Saunders Company. 1956.

Contents: Part I. *The Basic Mechanisms of Cardiac Symptoms and Their Management.* 1. Pathologic Physiology of Myocardial Insufficiency. 2. Digitalis. 3. Quinidine. 4. Arrhythmias. 5. Congestive Heart Failure. 6. Diet in Heart Disease. Part II. *Hypertensive Heart Disease and Arteriosclerotic Heart Disease.* 7. Hypertension and Hypertensive Heart Disease. 8. Arteriosclerotic Heart Disease. Part III. *Diseases of the Heart Secondary to Inflammation.* 9. Rheumatic Fever. 10. Subacute Bacterial Endocarditis. 11. Syphilitic Heart Disease. 12. Cor Pulmonale. 13. Non-specific Myocarditis. 14. Pericarditis. Part IV. *Congenital Heart Disease.* 15. Congenital Heart Disease. Part V. *Surgery in the Cardiac Patient.* 16. Anaesthesia in Heart Disease. 17. Surgery in Heart Disease. 18. Pregnancy in Heart Disease. 19. Cardiac Trauma. Part VI. *Diseases of the Heart Secondary to Metabolic Disorders.* 20. Hyperthyroidism and Heart Disease. 21. Hypothyroidism and Heart Disease. 22. Beriberi Heart Disease. Part VII. *Emotions, Adjustments, and Rehabilitation in Heart Disease.* 23. Psychosomatic Aspects of Heart Disease. 24. Living with a Sick Heart. 25. Rehabilitation of the Cardiac Patient. Appendix. Diets, Menus, Recipes, and Table of Sodium and Potassium Contents. Index.

Correct treatment of any medical disease requires a sound knowledge of the basic pathogenic mechanisms. Therapy directed against the existing disease-state is much more likely to be successful than when the diagnosis is faulty. These remarks may be trite, but they are not infrequently ignored by authors of books on medical treatment. The writers of this book have not fallen into this trap and as a result they have produced an acceptable book on the treatment of heart disease. They adopt a 'clinico-physiologic approach' and do not hesitate to discuss criteria of diagnosis in considerable detail. Inevitably it has become much more than a book on treatment; there are numerous electrocardiographs, X-rays and diagrams to illustrate the principles being discussed.

There is, of course, much on therapy, practically all of it sound and acceptable. The authors do not hesitate to express their own views on controversial matters. In fact one rather gets a bit too much of statements 'based on their own extensive experience'!

But it is a fault in the right direction. It is really surprising to find how much one can say on this subject and there is not a great deal of padding in these 549 pages. Each chapter includes a large number of (almost entirely American) references and there is an adequate summary.

One can easily quibble at small matters. The metric system is used extensively but every now and then a dose is given in grains. Proprietary names of drugs are frequently mentioned (sometimes coupled with the pharmacopeal name, sometimes not). The amount of space devoted to the fat content of foods is small in comparison with that devoted to its salt content. This is contrary to modern trends and unexpected in a book as up to date as this one undoubtedly is. The authors fight shy of long-term anticoagulant therapy on account of the high morbidity and mortality possibly associated with this treatment (this may be because they continue to use dicoumarol). Their advice in favour of ligation of veins in pulmonary embolism is also against the modern trend. This is surely a rare operation nowadays. But these, and other minor criticisms, do not detract from the value of this book. It is as clear and lucid a description of modern treatment in heart disease as one can find anywhere.

C.M.

TWO BIOGRAPHIES

Life of Sir John Bland Sutton, Bt. 1855-1936. By W. R. Bett, M.R.C.S. Eng. Foreword by Lord Webb-Johnson. Pp. viii + 100. Plates 5. 20s. net. Edinburgh and London: E. & S. Livingstone Ltd. 1956.

Contents: Foreword. Preface. I. Rough-and-Ready Beginnings. II. The Middlesex Hospital. III. Gynaecological Surgery: A Stormy Chapter. IV. The Royal College of Surgeons of England. V. Among the Dentists. VI. The Private Lives of Public Friends. VII. Wand'ring from Clime to Clime. VIII. 'Those Friends Thou Hast'. IX. 'Books, The Children of the Brain'. X. 'I am Seeking a Man'.

Sir William Arbuthnot Lane, Bt. C.B., M.S. An enquiry into the mind and influence of a surgeon. By T. B. Layton. Pp. viii + 128. 21s. net + 10d. postage abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1956.

Contents: Preface. I. 'Willie': Origins and Education. II. Guy's. III. Examinations. IV. Qualified. V. The Wonderful Hands. VI. The Perfectionist. VII. Back at Guy's. VIII Marriage. IX. The Early Nineties. X. Cleft Palate. XI. The Role of the Nursing Profession in Technique. XII. Fractures. XIII. More about Technique. XIV. Illness and Relaxations. XV. Constipation. XVI. Mechnikov. XVII. The Reaction of Lane's Views. XVIII. The Great Debate. XIX. House Surgeons' Stories. XX. War. XXI. The Americas. XXII. Great Ormond Street, New Health Society and Other Activities. Index.

These two books in the series of notable historical biographies from the House of Livingstone, deal with two colourful surgeons who flourished in London around the turn of the century. Sir William Arbuthnot Lane, Bart. (1856-1943), known as 'Willie', lived and worked in the atmosphere of Guy's Hospital. The name of Sir John Bland-Sutton (1855-1936), on the other hand, is inextricably bound up with the Middlesex Hospital. Their lives mirror to a large extent the doings and happenings of the London medical (or should it be surgical?) world and their respective hospitals.

They both had a link with South Africa. Lane was barely one month old when he arrived with his parents; his father's regiment had been ordered to South Africa for the '1st Kaffir War'. They moved on to Bengal when Lane was 14 months old. Bland-Sutton delivered the inaugural address at the initiation of C. F. M. Saint as the Professor of Surgery in Cape Town! He suggested to the new professor the wisdom of limiting a lecture to 30 or 40 minutes, save in exceptional circumstances! (one seed which did *not* fall on fertile ground!)

Both were pioneers born at a time to be able to benefit from the full flood of surgical progress made possible by anaesthesia and antiseptics. Their achievements are well known, so are many of their mistakes. Lane, the perfectionist, with his no-touch technique, which made possible in those gloveless days the successful treatment of simple fractures by converting them into compound fractures, and the application of an 'internal splint'; his insistence of the importance of a functional result in fractures and the care he took to ensure this; his pioneering efforts in the treatment of empyaema and cleft palate; these have all been largely forgotten—his 'kinks' and his operation of colectomy for constipation are always remembered. 'Stasis' and 'auto-intoxication' are rarely mentioned nowadays. It is not surprising that he made mistakes, when opening up 'operative fields unthought of'.

Bland-Sutton was called a criminal mutilator of women for his pioneer work on gynaecologic surgery. The Chelsea Hospital, where he did much of this work, became the Mecca for pelvic surgeons from all over the world. His operations 'disestablished the couch invalid', generally a woman with fibroids or an ovarian tumour or the sequelae of pelvic inflamation. She could become a companion to her husband, or earn her own living and cease to be a domestic incubus. He also pioneered much of what we now know of odontomes and other dental conditions. In addition to this, he found comparative anatomy to be another fascinating field. He studied the lives and illnesses of animals in the zoo with almost as much enthusiasm as he devoted to human medicine.

These two pioneers of the late 19th and early 20th centuries live again in these books. Most of their early contemporaries have died as have many of the later ones. The records of a large number of fascinating incidents of these times will have perished with them. For this reason we should be grateful to both the authors of these biographies for recording their memories before it is too late.

C.M

LAUGHTER AND HUMOUR

Laughter and the Sense of Humour. By Edmund Bergler, M.D. Pp. xii + 297. 85.00. New York: Intercontinental Medical Book Corporation in cooperation with Grune & Stratton, Inc. 1956.

Contents: Foreword. 1. Ephemeral Theories—Eternal Laughter. 2. The Three Triads of the Individual Pre-History of Laughter. 3. Laughter in the Adult Sense: Internal Antidote against Fear of One's Own Psychic Masochism. 4. Wit: The Intellectual High Point of All Adult Laughing Matter. 5. The Four Pillars of That 'Mysterious' Sense of Humor: Wit, the Comic, Self-Derision, Jokes in Lieu of Sympathy. 6. Irony, Sarcasm, 'Life's Little Ironies', Cynicism. Repartee. 7. Two Fascinating Theories on Laughter Regretfully Disputed. 8. Anti-Pompousness: The Basis of the American Sense of Humor. 9. Fun-Deficiency: The Bores and the Bored. 10. Corrupted Laughter—Anti-New, 'Directed,' and Prostituted. 11. Ego's Dread Lest the Internal Wit-Tables Be Turned. 12. Nothing New in the World of Wit. 13. Creation of the Artificial Victim in Laughter.

Dr. Bergler is a well-known member of various psychoanalytic and psychiatric societies and has 16 books and 225 scientific papers to his credit. In this volume he analyses what laughter is, what a sense of humour is, what a joke is, and why it is a joke. Dr. Bergler traces the various theories on laughter from the time of Plato with his pleasure-pain theory, through the Italian Renaissance theories of laughter to Hobbes's famous sentence 'Laughter is nothing but the sudden glory arising from some sudden conception of some eminence in ourselves; by comparison with the inferiority of others, or with our own formerly'. He discusses the theories of many other philosophers including Descartes, Hobbes, Spinoza, Samuel Johnson, Kant and Schopenhauer, as well as the five components of the sense of humour—wit, the comic, selfderision, grim humour, jokes in lieu of sympathy. The average reader in search of a book on jokes will be disappointed even though numerous examples of puns, witticisms, fun deficiency, and the 'Ego's dread lest the internal wit-tables be turned' are given. In his final paragraph the author says that a critical reviewer of one of his books stated that 'an author who wants to be understood must come down to the level of his readers.' This is the opinion of the present reviewer, who found this scientific work would scarcely be sought by the average reader but was worth the study of those interested in the whole concept of the superego, psychic masochism and double inner defences. For such this volume will find a place on their shelves.

C.D.B.

PRACTICAL UROLOGY

Practical Urology: Case-Comments and Late Results. By Alex. E. Roche, M.A., M.D., M.Ch. (Camb.), F.R.C.S. (Eng.). Pp. xii + 258 + 132 illustrations. £1 15s. 0d. net. London: H. K. Lewis & Co., Ltd. 1956.

Contents: Preface. Abbreviations. Case 1. Removal of stones from posterior urethra, bladder, and urethral pouch. No re-formation of stones there in 21 years. 5. Excision of large R. ureterocele. 44 stones removed from it and R. ureter. No R. ureteric stones 20 years later. 10. L. renal neoplasm in girl aged 6, well 18½ years after L. nephrectomy. Lessons from 5 renal neoplasms in 1936. 15. R. hydronephrosis (8¼ lb) causing oedema and cynoasis of flanks and lower limbs by venous pressure. Patient well nearly 15 years after R. nephrectomy. 20. Calcified large L. hypernephroma, present 5½ years, removed (with hypernephromatous lymph gland) from boy of 19, well 14 years later. Consideration of calcification in renal neoplasms. 25. Nephrectomy for R. renal neoplasm. Patient well 10½ years later. 30. Multiple R. renal stones with malignant papilloma. Recurrent bladder growths. Survival after R. nephrectomy for over 3 years. 35. Large bladder stone

with pyelonephritis. Snap diagnosis 'Influenza'! 40. Large stone in R. ureterocele, with hydro-ureter and hydronephrosis. Retzius drainage. 45. Huge bladder and other stones. Uraemic death. 50. Large bilateral low ureteric stones removed from man of 43, with also L. renal staghorn stone, incompletely removed later. Originally no I.V.P. evidence of R. renal function; now none of L.! Index.

Mr. Roche tackles his subject in a rather unusual way in this book, not grouping the cases belonging to one condition under one heading, but relating each case separately, and giving history, findings, diagnosis, treatment and late results only in so far as they applied to this particular case. Each case is listed separately in the Contents, and at the head of this review only one-fifth of the long list is reproduced.

It is an arduous task to read through an ordinary text-book, unless one is studying for an examination, or looking up a particular point. But when the subject matter is presented in this novel way, it is really quite good entertainment, and makes easy reading to go through the case histories of 3 or 4 cases in an evening.

The index at the back makes it possible to look up a particular type of case that one might be dealing with at the time.

The X-ray reproductions are in most cases excellent, and bring out the diagnostic points rather well.

It would have been a good idea not to mention the diagnosis of each case on the top of the page, so that the reader might have tried to make the diagnosis for himself as he reads, and as he looked at the X-rays, and then check up on his own opinion at the end of each record. The index or list of contents would still have served for looking up the details of any particular condition one might be wishing to refer to.

This book can be recommended to all those who are likely to handle urological cases, whether as urologist, general surgeon or general practitioner.

J.D.J.

EMBRYOLOGY

Aids to Embryology. Fifth Edition. By J. S. Baxter, M.A., M.Sc., M.D., F.R.C.S. Pp. viii + 196 with 61 illustrations. 8s. 6d. London: Baillière, Tindall and Cox Ltd. 1956.

Contents: Introduction. I. The Germ Cells. II. Segmentation and Germ Layer Formation. III. Changes in the Female Genital Tract. IV. Implantation and Placentation. V. Formation of the Embryo. Determination of Age. VI. The Skin and its Accessory Structures. VII. The Nervous System. VIII. Development of the Organs of Special Sense. IX. The Alimentary Canal and Related Structures. X. The Circulatory System. XI. The Coelomic Cavities. XII. The Urogenital System. XIII. The Muscular and Skeletal Systems. XIV. The Transmission of Hereditary Characters. Index.

Embryology is one of the medical sciences for the study of which there is a special need of a short authoritative book such as is provided by this volume in the Students' Aid Series. Incorporated in anatomical teaching for a long time as a scientific 'frill', developmental anatomy has only recently become regarded as an important branch of that subject.

It still has to be recognized also that Embryology is more a pure than an applied science, more intellectually satisfying than utilitarian. Consequently its claims on the student's attention and on academic time cannot be overindulged at the expense of the basic medical subjects.

This is where a book that can impart the maximum of information in the minimum of time has an assured place. That is the value of this handy volume, in which Professor J. S. Baxter has succeeded in giving a concise yet very complete text-book account of the development of the foetal membranes and of all the systems and organs of the human body.

Whether it is a sound knowledge of Embryology that is required for examination purposes, or whether it is just a matter of refreshing old knowledge or satisfying scientific curiosity, this book can be confidently recommended. It is, moreover, very good value at the present price.

The author acknowledges some suggestions by reviewers of previous editions. The present reviewer feels that this book would be pedagogically improved if, when first encountered in the text, all standard embryological terms such as ectoderm, entoderm, mesoderm, trophoblast, primitive streak, intermediate cell mass, septum primum etc. were given emphasis by typographical differentiation.

M.R.D.

ADVANCES IN PHARMACOLOGY

Recent Advances in Pharmacology. Second Edition. J. M. Robson, M.D., D.Sc., F.R.S.E., and C. A. Keele, M.D., F.R.C.P. (Lond.). Pp. xii + 501. 66 Illustrations. 40s. London: J. & A. Churchill Ltd. 1956.

Contents: 1. Noradrenaline, Adrenaline and Isoprenaline. 2. Neuromuscular Blocking Drugs. 3. Hypotensive Drugs. 4. Histamine and 5-Hydroxytryptamine. 5. Chlorpromazine and Anti-emetics. 6. The Adreno-Hypophyseal System. 7. The Newer Antibiotics. 8. The Chemotherapy of Tuberculosis. 9. Salicylates-Phenylbutazone-Drugs in Gout. 10. Nucleotoxic Drugs. 11. Radiation Hazards. 12. Hemopoietic Substances. 13. Anticoagulant Drugs. 14. The Control of Body Functions by Chemical Substances. Index.

This edition is essentially a new book with extensive revision of the topics which appeared in the first edition and with information on adrenaline and noradrenaline, hypotensive drugs, histamine and 5-hydroxytryptamine, chlorpromazine, aldosterone, the chemotherapy of tuberculosis, salicylates, phenylbutazone and drugs in gout, nucleotoxic drugs, radiation hazards, cyanocobalamin and other haemopoietic substances. There are 66 illustrations, several tables, and numerous chemical formulae. Every chapter has many references.

The authors have produced a valuable volume for the clinical and laboratory investigator and for those in general practice interested in 'academic' scientific developments and a basic understanding of our knowledge of certain newer drugs. As examples of some of the contents may be mentioned the estimation of adrenaline and noradrenaline in blood, urine and tissue extracts, the formation of these catecholamines and their actions in the body, the factors influencing their excretion, and the significance of all this in the symptomatology and diagnosis of phaeochromocytoma. Recent developments concerning neuromuscular blocking drugs used as muscular relaxants and their antagonists are reviewed. In the section dealing with hypotensive drugs the actions and uses of ganglion-blocking agents, and other drugs such as rauwolfia and veratrum alkaloids and hydralazine—all used empirically in essential hypertension—are described. There is systematic and well-presented information on the enigmatic physiological substance histamine, on which so much is always being written, and on 5-hydroxytryptamine, on which also so much work is being done. Laboratory workers will be interested in the information on tests for these substances and many others considered in the book, for example the control of anticoagulant therapy. The modern investigations on salicylates which have led to better understanding of their effects on metabolism and respiration and new views on their mode of action in rheumatic fever have been given special emphasis.

The complicated ramifications of pharmacology and the remarkable advances in many fields of laboratory and clinical research are well demonstrated in this book, which can be recommended to all who desire to follow recent developments in our knowledge of drugs.

N.S.

CORRESPONDENCE : BRIEWERUBRIEK

'MEDICAL REPRESENTATIVES'

To the Editor: While, like the majority of doctors, I am prepared to interview medical representatives, and am on good terms with most of them, we must be clear about what Mr. Ben Kessel[1] means when he says that a representative is 'an expert on these preparations to advise the physician.' If the representative is willing (as most of them are) to advise us that such-and-such a preparation is available, and (if he wishes) to draw our attention to clinical trial and other evidence of their use, we have no quarrel with him. When, however, he begins to assume a medical knowledge which he has not got then he begins to bore. He also becomes more and more boring to me as he persists in pushing a product for which I obviously and frankly have no use. His real function is not so

much the scientific and therapeutic education of the medical profession as the selling of his goods. At any rate the management of the pharmaceutical firms assess his value to them not on his clinical acumen but on his salesmanship—the results he gets. Every 'rep' knows that quite well. He has to make his living, and the medical profession should (and on the whole does) treat him with consideration. My one grouch is that he is taught to enter my surgery with great *bonhomie* extending his hand for a hearty handshake. For the information of the reps, that puts me off. After all, it is the host—not the guest—who sets the style of greeting. And I am one of those who dislike being pounced on with an extended hand. Moreover, the more sales talk, laudation of his goods and extrovert Carnegie stuff I get the more sales-resistant I become. I also dislike being cross-questioned on my use of the firm's products, and having my prescriptions checked by the 'rep' at the chemist's. My prescriptions are my business, not the rep's. Most representatives come in to do business and get it over expeditiously; one or two, however, are time-wasting chatterboxes.

Furthermore: if there is anything a doctor dislikes more than a curbside consultation it is wayside salesmanship—in the passage, on the street, in the lift. After all, we *are* entitled to a little social life and personal freedom from being medically chased on every occasion we are spotted and pounced on outside our hours.

Frank Proksch

26 High Road
Durban
26 September 1956

1. Kessel, B. (1956): S. Afr. Med. J., **30**, 928.

BLOOD APARTHEID

To the Editor: While Federal Council met in Cape Town last week it was announced in the daily press that the Southern Transvaal Branch of the Association had telegraphed a resolution on blood *apartheid* to them and that they were discussing the matter.

We anxiously await a statement from Federal Council on the contents of the telegram and on what action they themselves have decided to take.

While I do not wish to raise political matters in the *Journal*, it must be pointed out that the daily press of all shades of political opinion had incomplete and often misleading so-called 'scientific' statements on this subject prominently displayed in its news columns on several occasions in the last week. These statements have raised many doubts in the minds of the public and possibly unnecessary fears of the results of blood transfusion.

We as doctors and scientists cannot allow ourselves to be swayed by political or emotional side-issues in this or in any other matter of medical importance. If Federal Council feel that there are or are not valid scientific grounds for blood *apartheid* they, as the official mouthpiece of the Association, should let the public know where we stand, at once and in no uncertain terms. Only in this way can we show that we maintain our intellectual and scientific honesty as medical men.

R. L. Kleinman

4 Dunrobin Flats
Kloof Road
Sea Point, Cape
7 October 1956

[At the recent meeting of the Federal Council it was resolved to publish no statement on this subject other than that the matter was under consideration and receiving attention.—*Editor.*]

RACIAL DISTINCTIONS

To the Editor: The issue of the *Journal* of 6 October 1956 brings to the attention of the profession two alarming items. In the report of the meeting of the South African Medical and Dental Council[1] are included the facts that the Transvaal Provincial Administration pays non-White doctors lower salaries than White, and that the bloods of non-White and White donors to blood transfusion services are to be marked and kept separately.

It is difficult and indeed dangerous to raise political subjects in the *Journal* and in the Association, but one feels that there is a difference between taking sides on sociological attitudes towards *apartheid* and the attempt by party politicians to infuse science with unfounded racial theories. Similarly it is a new idea that doctors of different skin colours—not different qualifications and responsibilities—should receive different rates of pay merely because of that colour.

Either the Medical Association should appoint a vigilance committee to keep an eye on these matters or an independent body of practitioners should be formed to do so.

Party politics are all very well—but medicine in South Africa both as a profession and a science must see that it is not led the way of the profession in Nazi Germany when it was dominated by the racial mythology of the Nuremberg laws.

A. A. Zabow

309 Voortrekker Road
Maitland, Cape
6 October 1956

1. Meeting of S. Afr. Med. Dent. Coun. (1956): S. Afr. Med. J., **30**, 968.

OPKNAPPINGSKURSUS (REFRESHER COURSE) VIR ALGEMENE PRAKTISYNS, DURBAN

Aan die Redakteur: Ek het onlangs die voorreg gehad om die kursus in Durban by te woon. Graag wil ek die Natalse mediese skool gelukwens met die interessante en leersame kursus wat aangebied was. Soggens het ons na lesings geluister, terwyl daar elke middag kliniese demonstrasies en saalrondtes was. Dit was veral op hierdie gebied dat die staf presteer het, aangesien daar elke middag 'n keuse van minstens twee of drie programme was. Ek kan met die grootste vrymoedigheid my mede-kollegas aanraai om in die toekoms hierdie kursus by te woon.

T. B. de Bruyn

Cradockstraat 4
Steynsburg
7 Oktober 1956

ECONOMIC PROBLEMS FOR MEDICAL PRACTITIONERS

To the Editor: In the *Journal* of 29 September 1956 Dr. J. H. Struthers[1] writes: ' . . . . it may be that serious practical and economic problems are going to arise from the possible overproduction of specialists.'

This, in my view, is an understatement as far as both general practitioners and specialists are concerned, if they practise in cities, and if they are dependent on private practice, i.e. on fee-paying patients.

My estimate is that it will be found that there is one private doctor per 300 private fee-paying patients in the cities, and that there are hundreds of doctors in private practice who earn less than £1,200 a year. If this estimate is supported by the facts, then a message should go out to parents warning them that their children will not be able to make ends meet if they choose medicine as a profession.

'Durban'

Durban
8 October 1956

1. Struthers, J. H. (1956): **30**, 946.

M.B., CH.B. (U.C.T.)

Aan die Redakteur: Die afgelope jare is ek dikwels deur pasiënte gevra waarvoor die mediese graad U.C.T. staan. Sommige verkeer selfs onder die indruk dat dit een of ander spesialis-graad is. Ek wonder dus of dit nie beter (miskien moet ek liewers sê regverdiger), sal wees as die graduandi van Kaapstad liewers M.B., Ch.B. (Kaap) of (Cape), i.p.v. M.B., Ch.B. (U.C.T.), sal skryf nie.

T. B. de Bruyn

Cradockstraat 4
Steynsburg
7 Oktober 1956

[Die Suid-Afrikaanse Mediese en Tandheelkundige Raad gebruik die skryftrant M.B., Ch.B. (Univ. Cape Town), of M.B., Ch.B. (Univ. Kaapstad). Dit word algemeen geskryf met die weglating van ‚Univ'.—*Red.*]